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## THE VALUE OF THE COLD PRESSOR TEST IN THE PREDICTION OF HYPERTENSION AND TOXÆMIA IN PREGNANCY.[1]

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MANY divergent opinions have been expressed about the value of the cold pressor test for the prediction of hypertension in pregnancy since the first description of this test by Hines and Brown in 1932.

It has long been known that the vasomotor centre can be excited by many factors, such as stimulation of sensory nerves by cold, pain, electrical or emotional disturbances. A number of methods have been devised in attempts to single out persons who will develop persistent hypertension later in life. In these methods the aim has been to increase the blood pressure in patients who are suspected of having a labile vasomotor mechanism by means of a "standard stimulus". In the cold pressor test, Hines and Brown[1] have used water at 4° C. for this "standard stimulus". They found that the normal person responded to the stimulus by a slight rise in both the systolic and diastolic blood pressures with a rapid fall to normal three minutes after removal of the stimulus. In the hypertensive person a much greater and more prolonged rise in blood pressure was obtained. The response to the stimulus occurred too rapidly to be due to hormonal or chemical factors. Hines and Brown feel that it must have a neurogenic basis and advance as proof of this hypothesis the evidence that the response has been obtained after bilateral suprarenalectomy in dogs.

[1] This work was made possible by a grant from the National Health and Medical Research Council of Australia.

### The Ice Pressor Test as Described by Hines and Brown.

The patient is rested for twenty minutes and the basal blood pressure is taken. A sphygmomanometer cuff is placed on one arm and the opposite hand is completely immersed in water at 4° C. Blood pressure readings are taken at intervals of thirty and sixty seconds after immersion. The highest reading is called the index of response. The hand is then removed from the ice water and blood pressure readings are taken at intervals of two minutes until the basal level is again reached. For reliable results to be obtained with this test it is necessary to determine the true basal blood pressure; hence readings must be repeated until a constant value is reached before the investigator proceeds with the later part of the test. The patient is described as a hyperreactor if in response to the cold stimulus the systolic blood pressure rises above the basal level by more than 20 millimetres of mercury or the diastolic pressure by more than 15 millimetres. The return of the blood pressure to the basal level is delayed in patients with established hypertension. If the maximal or "ceiling" pressure is greater than 140 millimetres of mercury (systolic) and 90 millimetres of mercury (diastolic), the patient is even more certain to have a hyperreactive vasoconstrictor mechanism.

### Review of the Literature on the Value of the Ice Pressor Test in Pregnancy.

Since 1932 a number of papers have given detailed accounts of the application of the ice pressor test for the prediction of hypertension in pregnant women; but the various authors have arrived at different conclusions as to its value. Reid and Teel[2] and also Chesley and Chesley[3] conclude that the test fails to distinguish hypertensive from other types of toxæmia, and also that it gives no warning of impending toxæmia. They found that the response of the same patient to repeated tests was

Table I.

| Average Basal Blood Pressure. (Millimetres of Mercury.) | Type of Pregnancy. | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Normal. | | Mildly Toxæmic. | | Preeclamptic. | | Hypertensive. | |
| | Number of Patients. | Number of Readings. | Number of Patients. | Number of Readings. | Number of Patients. | Number of Readings. | Number of Patients. | Number of Readings. |
| **Systolic:** | | | | | | | | |
| Second month | 116 | 23 | — | — | 117 | 4 | 130 | 9 |
| Third month | 112 | 32 | — | — | 124 | 2 | 135 | 18 |
| Fourth month | 115 | 49 | 120 | 3 | 112 | 5 | 134 | 29 |
| Fifth month | 111 | 43 | 119 | 3 | 131 | 10 | 137 | 41 |
| Sixth month | 114 | 71 | 110 | 2 | 126 | 13 | 140 | 57 |
| Seventh month | 115 | 73 | 114 | 4 | 141 | 16 | 161 | 54 |
| Eighth month | 118 | 84 | 118 | 7 | 155 | 17 | 143 | 58 |
| Ninth month | 133 | 88 | 134 | 7 | 162 | 10 | 171 | 66 |
| **Diastolic:** | | | | | | | | |
| Second month | 74 | 23 | 80 | 2 | 79 | 4 | 95 | 9 |
| Third month | 69 | 33 | 80 | 3 | 84 | 2 | 83 | 19 |
| Fourth month | 71 | 49 | 82 | 3 | 75 | 5 | 83 | 28 |
| Fifth month | 70 | 43 | 78 | 2 | 85 | 10 | 84 | 39 |
| Sixth month | 70 | 71 | 82 | 2 | 81 | 13 | 90 | 52 |
| Seventh month | 72 | 72 | 74 | 6 | 88 | 14 | 91 | 54 |
| Eighth month | 78 | 80 | 84 | 5 | 103 | 14 | 91 | 56 |
| Ninth month | 98 | 48 | 95 | 4 | 105 | 8 | 103 | 59 |

not constant and therefore considered the test to be unreliable.

Dieckmann and his collaborators,[4] Randall, Murray and Mussey,[5] Briggs and Oerting,[6] Browne,[7] and Bak[8] all found that hypertension and often toxæmia developed more frequently in the later stages of the pregnancy of hyperreactors to the cold pressor test than in the pregnancy of women giving a normal response to the test. Dieckmann *et alii*[4] found that the mean ante-partum rise in the systolic blood pressure increased from 20 millimetres of mercury in cold pressor tests on normal pregnant women with no family history of hypertension to 36 millimetres of mercury in tests on women with a family history of hypertension. Although the mean ante-partum rise in the systolic blood pressure in cold pressor tests on eclamptic patients was only slightly higher than that found in normal pregnant women, a much greater rise, namely, 52 millimetres of mercury, was observed in tests on hypertensive patients. These workers found that the test gave constant results when applied to the same patient in their series of at least two tests on each of 500 women. In some instances they were able to reproduce their results as many as eight times in an individual case.

Randall, Murray and Mussey,[5] after testing 104 pregnant patients, concluded that toxæmia was unlikely to develop when a persistently normal reaction was obtained. All their patients who developed toxæmia were hyperreactors.

Briggs and Oerting[6] used the cold pressor test on 233 pregnant women and found that 32 were hyperreactors. These patients were interesting from another point of view, namely, that of heredity. Both parents of each of ten patients were hypertensive, and all the patients themselves were hyperreactors. Nine of them also developed toxæmia during their pregnancies. In a second group one parent of each of the 44 patients was hypertensive and 13 of these women were hyperreactors to the cold stimulus. Three of the 13 patients became toxæmic before the termination of their pregnancies. None of the parents of the remaining 179 women were hyperreactors, and all but two patients in this group gave a normal response to the cold pressor test.

Bak[8] found that those patients who developed toxæmia in a group of 110 pregnant women tested for this cold response had all been hyperreactors.

If it was true that the cold pressor test was capable of showing the presence of a hyperreactive vasoconstrictor mechanism in women prior to or early in pregnancy, and also that such women frequently developed hypertension and sometimes toxæmia later in pregnancy, this test would be a valuable aid to the obstetrician.

Table II.

*Ceiling Blood Pressure Revealed by Ice Pressor Test.*

| Ceiling Blood Pressure. (Millimetres of Mercury.) | Type of Pregnancy. | | | | | |
|---|---|---|---|---|---|---|
| | Normal. | | Preeclamptic. | | Hypertensive. | |
| | Number of Patients. | Number of Readings. | Number of Patients. | Number of Readings. | Number of Patients. | Number of Readings. |
| **Systolic:** | | | | | | |
| Second month | 140 | 29 | 147 | 2 | 143 | 11 |
| Third month | 135 | 34 | 123 | 2 | 146 | 12 |
| Fourth month | 137 | 43 | 132 | 5 | 150 | 17 |
| Fifth month | 131 | 44 | 142 | 11 | 145 | 18 |
| Sixth month | 142 | 68 | 145 | 11 | 152 | 29 |
| Seventh month | 131 | 63 | 148 | 12 | 152 | 30 |
| Eighth month | 129 | 67 | 144 | 3 | 150 | 33 |
| Ninth month | 148 | 12 | 175 | 3 | 160 | 2 |
| **Diastolic:** | | | | | | |
| Second month | 95 | 29 | 107 | 2 | 103 | 11 |
| Third month | 94 | 34 | 92 | 2 | 106 | 12 |
| Fourth month | 93 | 33 | 103 | 5 | 112 | 16 |
| Fifth month | 98 | 44 | 108 | 11 | 104 | 18 |
| Sixth month | 94 | 68 | 106 | 11 | 104 | 29 |
| Seventh month | 97 | 67 | 113 | 12 | 102 | 30 |
| Eighth month | 104 | 12 | 113 | 3 | 112 | 32 |
| Ninth month | — | — | 137 | 3 | 130 | 1 |

We have attempted to obtain further evidence about this test by studying its effects on two groups of patients who provided entirely different material from that used by other workers. Firstly, there was a group of 215 women who had previously had a toxæmic pregnancy in which the blood pressure had been raised. These patients were available for study throughout a subsequent pregnancy. Secondly, we were able to use the cold pressor test on a large group of patients at two months and often also at twelve months after the termination of a toxæmic pregnancy.

### Experimental Method.

We followed Dieckmann's modification of the Hines and Brown test. There are three points of difference in these two methods: (i) In Dieckmann's method the sphygmomanometer cuff is attached to the arm of the hand immersed in the ice water instead of to the opposite arm. (ii) The water was kept at a temperature of 1° C., which could be maintained more easily than a temperature of 4° C. (iii) The hand was immersed in the ice water for two minutes instead of one minute. Our patients were rested in a comfortable room for at least forty-five minutes before the test was commenced, and several readings of the blood pressure were made before the result was recorded as the initial basal blood pressure.

TABLE III.

*A Comparison of the Incidence of Normal and Abnormal Responses to the Cold Pressor Test Two Months and Twelve Months after Normal and Toxæmic Pregnancy.*

| Type of Pregnancy. | Two Months After Pregnancy. (Number of Subjects.) | | Twelve Months After Pregnancy. (Number of Subjects.) | |
|---|---|---|---|---|
| | Normal Reaction. | Hyper-reaction. | Normal Reaction. | Hyper-reaction. |
| Normal .. .. | 27 | 6 | 14 | 2 |
| Mildly toxæmic .. | 22 | 8 | 14 | 3 |
| Preeclamptic and eclamptic .. | 112 | 117 | 76 | 58 |
| Hypertensive toxæmic .. | 44 | 89 | 32 | 27 |
| | 205 | 220 | 136 | 90 |
| Total .. .. | 425 | | 226 | |

### Preliminary Problems.

Certain questions of a general character had to be answered before the clinical value of the test could be assessed. Firstly, the conflicting statements of Reid and Teel[2] and of Chesley and Chesley[3] on the one hand, and of Dieckmann *et alii*[4] on the other, with regard to the constancy of response when a patient was tested repeatedly, had to be examined. Serial cold pressor tests were performed on 69 patients who were referred to us because of a previous toxæmic pregnancy. On 23 of them we were able to repeat the test as many as five to eight times.

Erratic results were obtained in the serial tests in only 12 of these instances. In all the other cases the results were either constant or of two types not previously recorded, neither of which could be termed erratic. The constant results were obtained in serial tests on 20 patients (see Figures I and II). The other types of result were obtained in the serial cold pressor tests on the remaining 37 patients. In one group all tests during the pregnancy produced a normal response to the cold stimulus except the last one, in which a hyperreaction occurred. Gradually increasing hyperrecation from early pregnancy to term was noted in the second group (see Figure III).

The results of cold pressor tests on 144 women investigated at two and twelve months after pregnancy were interesting. Constancy of results was observed in both tests on 106 patients. In some cases both tests produced a normal response, and in others both tests indicated hyperreaction. Another interesting observation was the persistent hyperreaction obtained in tests at two months after delivery, with a decline to a normal response in tests twelve months after delivery, in 27 other cases in this group. In the 11 remaining cases a normal response was obtained two months after delivery, but the response at twelve months was of the hyperreactive type (see Table IV).

A large series of tests has also provided interesting general information about variations in blood pressure levels during normal and abnormal pregnancies. There have been only a few studies on the normal blood pressure in pregnancy; but Dieckmann[9] correlated the results from several small series and found that all the data indicated that the systolic blood pressure dropped from five to ten millimetres of mercury below the level for normal non-pregnant women between the eighth and the thirty-second weeks of pregnancy; it then increased to the level of the normal non-pregnant woman as term approached. According to Robinson and Brucer,[10] in the normal person the range for systolic blood pressure is 90 to 120 millimetres of mercury and that for diastolic

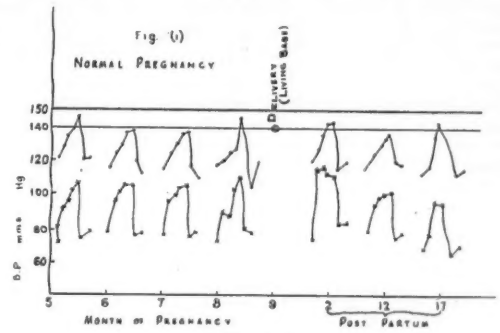


FIGURE I.

*Normal pregnancy.*

pressure is 60 to 80 millimetres of mercury, irrespective of age. Dieckmann and Kramer[9] showed that the systolic pressure during pregnancy lay between 90 and 119 millimetres of mercury and the diastolic pressure between 50 and 80 millimetres of mercury.

Browne[11] stresses the value of determining the basal blood pressure and states that if the systolic pressure is 130 millimetres of mercury or more and the diastolic pressure 70 millimetres of mercury or more, the patient is to be regarded as hypertensive.

We have been able to correlate the average basal blood pressure readings taken at monthly intervals during the pregnancies of 215 patients; 114 of these women had a normal pregnancy, 77 were classified as hypertensive, 17 as preeclamptic and seven as mildly toxæmic. The graphs (Figure IV), drawn from the average values recorded in Table I, show that our average blood pressure readings taken throughout normal pregnancies fall below Dieckmann's limits of 120 millimetres of mercury, systolic, and 80 millimetres of mercury, diastolic, except in the ninth month, when the average values for both systolic and diastolic pressures are raised. The few figures available for patients with mild toxæmia were similar to those from the normal patients.

In the case of the preeclamptic patients, however, the average systolic value rose to 130 millimetres of mercury in the fifth and sixth months and then steadily to an average of 160 millimetres of mercury at the ninth month. During the last three months of pregnancy the average diastolic reading rose steadily to 103 millimetres of mercury in these patients.

In the hypertensive toxæmic group the average systolic blood pressure exceeded Dieckmann's upper limit from the second month and increased sharply from the sixth

TABLE IV.

*A Correlation of Normal and Abnormal Responses to the Cold Pressor Test Two Months and Twelve Months after Delivery (the Same Patient).*

| Type of Pregnancy. | Normal Test Response Two and Twelve Months *Post Partum.* (Number of Patients.) | Hyperreaction Two and Twelve Months *Post Partum.* (Number of Patients.) | Hyperreaction at Two Months, Normal Response at Twelve Months *Post Partum.* (Number of Patients.) | Normal Response at Two Months, Hyperreaction at Twelve Months *Post Partum.* (Number of Patients.) | Total. |
|---|---|---|---|---|---|
| Normal | 4 | 0 | 2 | 1 | 7 |
| Mildly toxæmic | 3 | 0 | 7 | 1 | 11 |
| Preeclamptic and eclamptic | 34 | 35 | 11 | 8 | 88 |
| Hypertensive toxæmic | 13 | 17 | 7 | 1 | 38 |
| Total | 54 | 52 | 27 | 11 | 144 |

month onwards, finally reaching an average value of 170 millimetres of mercury at the ninth month. The average diastolic blood pressure in this group of patients was slightly greater than Dieckmann's upper limit of normal until the sixth month, when a further rise occurred, so that the diastolic pressure reached 103 millimetres of mercury at nine months. The moderate rise above the upper limits of normal in both systolic and diastolic blood pressures in the last month of pregnancy, even in patients whose pregnancy is normal, has not been mentioned by other workers.

The third preliminary problem is the question of what criteria indicate an abnormal response to the cold stimulus. Four factors have to be taken into consideration: (i) the basal blood pressure, (ii) the peak or ceiling blood pressure, (iii) the increase in blood pressure in response to the cold stimulus, (iv) the time taken for the ceiling

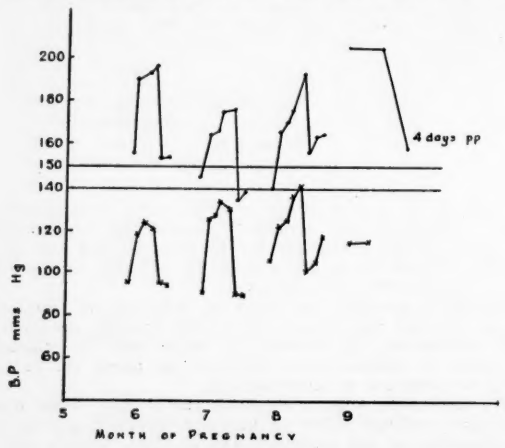


FIGURE II.
Hypertension (clinical classification).

value to fall to the basal blood pressure after removal of the cold stimulus. As has previously been mentioned, Hines and Brown[a] stress the importance of obtaining the true basal blood pressure, and hold that an increase in the systolic pressure of more than 22 millimetres of mercury is abnormal. They also state that the basal blood pressure should be attained two minutes after removal of the cold stimulus. Delay in return to the basal level is regarded as an indication of hyperreaction. They further suggest that a ceiling value of more than 140 millimetres of mercury, systolic, and 90 millimetres of mercury, diastolic, makes it certain that the patient has a hyperreactive vaso-constrictor mechanism. Dieckmann regards a higher rise above the basal level than that specified by Hines and Brown—namely, 30 millimetres of mercury—as necessary before a diagnosis can be made. A ceiling value of 145 millimetres of mercury systolic blood pressure is regarded as a definite indication of such a condition. He makes no statement regarding the significance of the time taken for the maximal reading to return to the basal level.

Browne[7] found that in patients whose ceiling systolic blood pressure value reached 150 millimetres of mercury or more, there was a high incidence of toxæmia. The time taken for the ceiling figure to return to the basal level was found to be valueless as an indication of the presence of hyperreaction in his patients.

From a general survey of the results of our serial tests on women with normal and toxæmic pregnancies, the diagnosis of hyperreaction was made after examination of (i) the basal blood pressure, (ii) the ceiling value, and (iii) the difference between basal and ceiling values. The time taken for the maximal blood pressure to return to the basal level was invariably short and proved useless as an indication of hyperreaction in patients whose basal blood pressure was normal or only slightly raised.

In the graphs (Figure V) drawn from the results recorded in Table II the average ceiling systolic and diastolic blood pressure readings obtained in response to

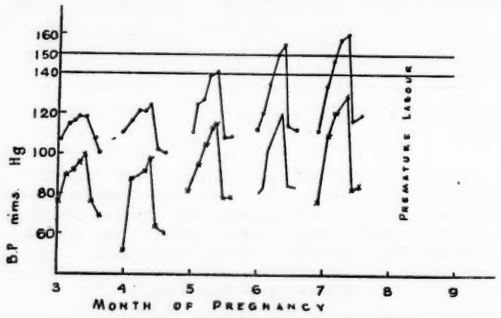


FIGURE III.
Hypertension (clinical classification).

cold pressor tests are compared with the upper limits suggested by Hines and Brown (145 millimetres of mercury, systolic, and 90 millimetres, diastolic). In normal pregnancy our results confirm the choice of 145 millimetres of mercury as the upper limit for the ceiling systolic blood pressure; however, the figure of 90 millimetres of mercury as the upper limit for the ceiling diastolic blood pressure appears to be too low, since our averages exceed this level throughout normal pregnancy. We suggest that 100 millimetres of mercury would be more suitable as the upper limit for the ceiling diastolic blood pressure during normal pregnancy.

The graphs also show that the upper limits of the ceiling systolic and diastolic blood pressures in normal pregnancy are exceeded in hypertensive patients right through the pregnancy and that both levels rise sharply during the last two months of pregnancy in these cases. In the preeclamptic patients the average ceiling diastolic blood pressures are similar to those observed in the hypertensive patients; but the average ceiling systolic pressures are somewhat lower, approximating to the upper limit observed in normal pregnancy until the ninth month, when a sharp

rise occurs. From consideration of our own results and of the conclusions of other workers, it was decided to assess the result of a test as indicating hyperreaction if a rise of at least 30 millimetres of mercury occurred in both the systolic and diastolic blood pressures above the basal levels. In addition, we have also assessed a test result as indicating hyperreaction if the ceiling value for the systolic blood pressure exceeded 145 millimetres of mercury and the diastolic pressure exceeded 100 millimetres of mercury, even if the difference between the basal and ceiling values was less than 30 millimetres of mercury. The reason for this was that we found a considerable group of results in which the ceiling value was at least 145 millimetres of mercury (systolic) and 100 millimetres (diastolic), and yet the difference between the basal and ceiling values was under 30 millimetres of mercury. Closer examination of these results showed that the basal blood pressure was frequently at the upper limit of normal or slightly raised. Further, the systolic and diastolic pressures three minutes after the removal of the cold stimulus were often lower than the basal levels. This raised the question whether the true basal level had really been obtained prior to application of the cold stimulus.

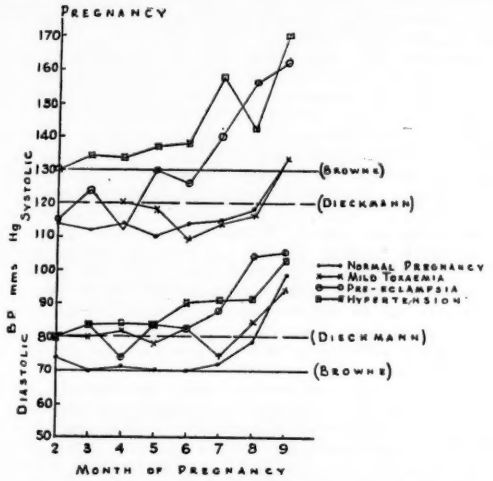


FIGURE IV.

Average basal blood pressures throughout normal and toxæmic pregnancy.

### Results of Cold Pressor Tests.

*During Pregnancy.*

In a series of 522 cold pressor tests performed during 200 pregnancies, all tests gave normal results in 84 instances, hyperreaction to the cold stimulus occurred in one test only on each of 31 patients and in more than one test on each of 85 patients. Only 13 of the patients, all of whose cold pressor tests gave normal results, developed hypertension in the later stages of their pregnancies. In four of these cases the hypertension was associated with preeclampsia. Half those patients, only one of whose tests gave a result of the hyperreactive type, had a normal pregnancy; but the other half developed either hypertensive toxæmia or preeclampsia. When mild hyperreaction occurred on more than one occasion, 26 of the 85 patients had a normal pregnancy, but 49 developed hypertensive toxæmia and 10 had preeclampsia.

Among the 71 apparently normal patients from whom repeated normal responses to the cold pressor test were obtained, the basal blood pressure of 26 was raised at delivery, and 15 of these gave a hyperreactive response two months later. Abnormal results were obtained on more than one occasion from 27 patients whose pregnancy appeared to be normal, and the basal blood pressure of 20 of these was raised at delivery. Sixteen of these patients were retested two months later and 13 of them gave a hyperreactive response.

Among 67 patients who developed hypertension during their pregnancies and 19 who developed preeclampsia, only nine of the first group and four of the second gave normal responses to ice pressor tests.

In many of these cases hyperreaction to the cold stimulus was observed for several months before the basal blood pressure became raised. Other patients had only a slight rise in the basal blood pressure, and hyperreaction to the cold stimulus provided confirmatory evidence of a sensitive vasomotor system. For those numerous patients whose systolic and diastolic blood pressures exceeded 150 and 100 millimetres of mercury respectively, the performance of the cold pressor test was of course both unnecessary and undesirable.

*Two Months After a Pregnancy in which Cold Pressor Tests were Performed.*

Persistent mild hyperreaction during normal pregnancy was usually associated with raised basal blood pressure at delivery and hyperreaction two months later. Of the patients clinically classified as hypertensive who were tested two months after delivery, three-quarters still either

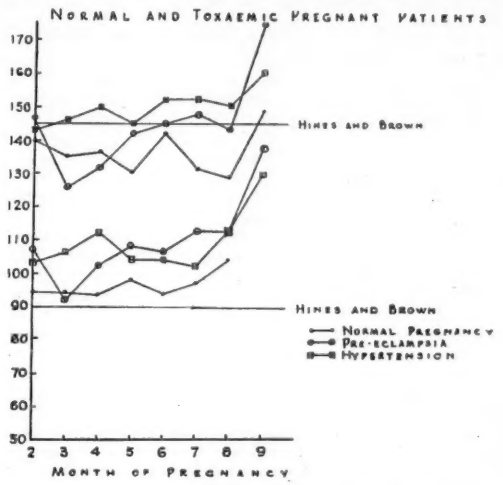


FIGURE V.

Average ceiling blood pressure reading in cold pressor tests on normal and toxæmic pregnant patients.

had a raised basal blood pressure or gave an abnormal response to the cold pressor test. The basal blood pressure was raised at delivery in all except two of the 22 patients whose pregnancy was complicated by preeclampsia; but it had dropped to normal in 12 of the 15 patients who were investigated two months later. However, hyperreaction to the cold stimulus was still obtained from six of these 12 patients. In three cases the basal blood pressure was still high two months *post partum*. Rather surprisingly it was found that after normal pregnancies in which all cold pressor tests produced normal responses, one-third of the patients gave a hyperreactive response two months after delivery.

*Two Months After Pregnancy in which No Cold Pressor Tests were Performed.*

In a group of 506 patients in which no cold pressor tests were made during the pregnancy, basal blood pressure readings and cold pressor tests were made two months after the completion of the pregnancy. In 126 of these cases the basal blood pressure was at least 150 millimetres of mercury, and in such cases no further evidence was necessary to demonstrate the existence of hypertension.

In a second group of 163 patients (32%) the basal systolic blood pressure ranged from 130 to 150 millimetres of mercury and the ceiling systolic and diastolic pressures

exceeded 145 and 100 millimetres of mercury respectively. The cold pressor test was thus useful in confirming the presence of hypertension in such borderline cases. In some of these cases the test also showed that the initial blood pressure reading was not the true basal level, since a lower reading was obtained after removal of the cold stimulus.

In a still larger proportion of cases (217 or 43%) the basal blood pressure was normal, but there was an abnormal difference between the basal and ceiling values, with the ceiling figures exceeding 145 and 100 millimetres of mercury respectively (systolic and diastolic). This showed that the person being tested was a potential hypertensive.

The incidence of hyperreaction after different types of pregnancy is interesting. An abnormal response followed normal pregnancy in 12% of this group of patients, but the figure increased to 36% when the patients had had mild toxæmia. After preeclampsia and eclampsia more than 50% of the patients had evidence of hypertension; 11% still had a basal systolic blood pressure above 150 millimetres of mercury, and another 44% gave an abnormal response to the cold pressor test. Of the patients who suffered from hypertensive toxæmia, 68% either had raised basal systolic blood pressure (25%) or gave an excessive response to the cold stimulus (43%).

*Two Months and Twelve Months After Delivery.*

A further point of interest is the assessment of the value of the cold pressor test at intervals after delivery, from the point of view of prognosis with regard to hypertension in later pregnancies. Tables III and IV present evidence showing that there is a high incidence of hyperreaction to a cold stimulus twelve months after pregnancy complicated by toxæmia or hypertension.

Table III shows that following the types of pregnancy studied, 51% of the patients had evidence of at least potential hypertension two months after delivery, and this was also true of 40% of the women tested twelve months after delivery. Table IV gives even more specific information about a smaller number of patients, each of whom was tested both two and twelve months after delivery. The interesting points brought out by this correlation are that many patients were either normal reactors or hyperreactors both two months and twelve months after delivery, but that about one-third of the total number who gave a hyperreaction at two months were apparently normal reactors at twelve months. More than half of the patients who were hyperreactors both at two and at twelve months after delivery showed a rise in their basal blood pressures only in the last few weeks of their pregnancy.

*The Appearance of Albuminuria After the Completion of Cold Pressor Tests.*

This large series of tests afforded the means of testing the report of Chesley, Markowitz and Wetchler,[12] in which it was stated that albumin was found in the urine of 23 hyperreactors after cold pressor tests, but that none was present in the urine of 24 normal reactors.

We tested the urine of 116 patients for albumin after completion of the cold pressor test. Twenty-five of them were hyperreactors and six showed a slight degree of albuminuria after the test, although the ceiling systolic blood pressure in the test did not exceed 170 millimetres of mercury in any instance. In five other instances the ceiling value exceeded 170 millimetres of mercury, but no albuminuria was detected. Our figures are not nearly so conclusive as those of Chesley, Markowitz and Wetchler.

## Discussion.

In our hands the cold pressor test has given consistent results throughout pregnancy in the majority of our cases, and erratic results have been the exception.

The view has been expressed generally in the literature that hypertension is due to constitutional factors; but our investigations seem to indicate that hyperreaction can develop during pregnancy and disappear again some time afterwards. We have observed a number of patients whose tests gave normal results until near term and then hyperreaction occurred. There is likewise a substantial group in which hyperreaction to the test was noted two months after pregnancy, but in which a normal response was obtained at twelve months. These results indicate that other than constitutional factors are involved.

Browne has suggested that a hypersensitivity of the vasomotor centre may be caused by either constitutional or environmental factors and that the latter may be toxic or infective agents. The observations just referred to would lend support to this suggestion.

From the practical point of view, what help do the results of the test give to the obstetrician? It should be realized, firstly, that the result of the test is no infallible answer to the question whether the patient is going to have a normal pregnancy or one complicated by hypertension or toxæmia. Some patients whose basal systolic blood pressure reaches 200 millimetres of mercury have completely normal pregnancies. Other patients, who give normal results to the test throughout pregnancy, occasionally develop hypertension or eclampsia; but this is the exception rather than the rule.

In general, patients who give a hyperreactive response to the test early in pregnancy develop hypertension as the pregnancy proceeds, and in some of these instances preeclampsia and eclampsia further complicate the pregnancy. To be forewarned of such possible complications is of value to the obstetrician.

Dieckmann has even held that it is possible to distinguish those patients who later become hypertensive from those who develop preeclampsia or eclampsia by means of this test. In our series some differences in the two groups were observed; for example, the average ceiling values for both systolic and diastolic blood pressures are above the normal level from early in pregnancy in the former group. This high figure was observed much later in pregnancy in many, but not in all, of the cases in which preeclampsia developed. In the hypertensive group there was a much greater number of patients whose monthly tests all produced a hyperreaction. On the whole, our results do not support the statement that these two groups of patients can be satisfactorily separated by the response to the cold pressor test. This may, of course, be due to preeclampsia developing in a patient who already has mild hypertension.

The results of cold pressor tests performed two and twelve months after delivery are valuable in assessing the prognosis of subsequent pregnancies. Those patients who still give hyperreactions twelve months after delivery should probably be classified as hypertensive. However, in the cases in which the hyperreaction response is replaced by a normal response some time after delivery, it can be assumed that the abnormal response during the present pregnancy was caused by some specific agent which may not be active in a subsequent pregnancy.

## Conclusion.

The results of serial cold pressor tests during pregnancy are of value to the obstetrician, since, even if only one response in the series is of the hyperreactive type, 50% of the patients giving such responses develop hypertensive or preeclamptic toxæmia. When two or more abnormal results are obtained, the number of patients who develop such toxæmia increases to nearly 70%. On the other hand, toxæmia occurs later in the pregnancy in only a few cases in which the response to the test is always normal.

Follow-up tests two and twelve months after delivery are sometimes helpful in assessing the prognosis of subsequent pregnancies, since patients who have hypertension or are likely to develop it can be differentiated from those unlikely to have this complication.

## Summary.

1. The cold pressor test of Hines and Brown is described and the literature on its application in pregnancy is reviewed.

2. The reliability of the test is discussed.

3. Variations in the average systolic and diastolic blood pressures during normal and toxæmic pregnancy are described.

4. Criteria for determining an abnormal response to the cold stimulus are suggested.

5. The results of serial cold pressor tests during normal and toxæmic pregnancy and of tests on the same patients two and twelve months after delivery are reported and discussed.

### References.


(1) E. A. Hines and G. E. Brown: "Hereditary Factor in Reaction of Blood Pressure to Standard Stimulus (Cold); Preliminary Report", *Proceedings of the Staff Meetings of the Mayo Clinic*, Volume X, 1935, page 371; quoted by W. J. Dieckmann, "The Toxemias of Pregnancy", 1941.

(2) D. E. Reid and H. M. Teel: "A Study of the 'Cold Test' in Normal and in Toxemic Pregnancy", *American Journal of Obstetrics and Gynecology*, Volume XXXV, 1938, page 305.

(3) L. C. Chesley and E. R. Chesley: "The Cold Pressor Test in Pregnancy", *Surgery, Gynecology and Obstetrics*, Volume LXIX, 1939, page 436.

(4) W. J. Dieckmann, M. L. Michel and P. W. Woodruff: "The Cold Pressor Test in Pregnancy", *American Journal of Obstetrics and Gynecology*, Volume XXXVI, 1938, page 408.

(5) L. M. Randall, S. E. Murray and R. D. Mussey: "The Cold Test in Pregnancy", *American Journal of Obstetrics and Gynecology*, Volume XXIX, 1935, page 362.

(6) J. F. Briggs and H. Oerting: "The Prognostic Value of the Cold Test in Pregnancy", *Minnesota Medicine*, Volume XX, 1937, page 382.

(7) F. J. Browne: "The Cold Pressor Test in Pregnancy", *The Journal of Obstetrics and Gynæcology of the British Empire*, Volume XLVII, 1940, page 365.

(8) M. Bak: "'*Kalt-Test*' *Untersuchungen an Schwangeren, Gebärenden und Wöchnerinnen*", *Monatsschrift für Geburtshülfe und Gynäkologie*, Volume CV, 1937, page 24.

(9) W. J. Dieckmann: "The Toxemias of Pregnancy", 1941, page 121.

(10) S. C. Robinson and M. Brucer: "Range of Normal Blood Pressure; a Statistical and Clinical Study of 11,383 Persons", *Archives of Internal Medicine*, Volume LXIV, 1939, page 409.

(11) F. J. Browne and G. H. Dodds: "Pregnancy in the Patient with Chronic Hypertension", *The Journal of Obstetrics and Gynæcology of the British Empire*, Volume XLIX, 1942, page 1.

(12) L. C. Chesley, I. Markowitz and B. B. Wetchler: *The Journal of Clinical Investigation*, Volume XVIII, 1939, page 51.


## UNDERGRADUATE AND POST-GRADUATE EDUCATION IN PHYSICAL MEDICINE.[1]


By HAROLD CRAWFORD, M.B., B.S., F.R.A.C.S.,
*Brisbane.*


WHEN the teaching of any subject is being considered there are certain factors that must be taken into account. First, we must define the subject to be taught and agree on the scope of the work covered by that subject. In the case of undergraduate teaching, unnecessary overloading of the curriculum must be avoided. We must decide how much instruction can be covered by the routine teaching in medicine, surgery *et cetera*, and how much special instruction is required.

### What is Physical Medicine?

In the same way as chemotherapy is the application of biochemistry to medicine, physical medicine is the application of biophysics in the diagnosis and treatment of abnormal and diseased conditions of the body. Biophysics covers a very wide and important field.

The Council on Physical Therapy of the American Medical Association has defined physical medicine as including the employment in the diagnosis and treatment of disease of the physical and other effective properties of light, heat, cold, water, electricity, massage manipulations, exercises and mechanical devices for physical and occupational therapy.

The definition is clear: physical medicine is the use of all physical means and instrumentation in the diagnosis and treatment of abnormal and diseased conditions of the body. According to this definition, the use of radiation from X rays and radium in diagnosis and treatment should be included; but this branch of the subject has advanced so rapidly that it has become an important specialty and has attracted many able physicians.

[1] Read at a meeting of the Australian Association of Physical Medicine on November 25, 1946, at Sydney.

### *Scope of the Work.*

The scope of physical medicine is wide and has been greatly increased as the result of the recent war. Dr. Frank Ober, professor of orthopædics and assistant dean of the Harvard Medical School,[1] has made the following statement: "The last war established orthopædic surgery as a recognised specialty; this war may well do the same for physical medicine."

In the Tomlinson report on the rehabilitation and resettlement of disabled persons, published in Great Britain in January, 1943, the importance of physical medicine in such rehabilitation was stressed. In America, the report of the Baruch Committee on Physical Medicine stressed the need for the development of physical medicine. After the issuing of this report Mr. Baruch gave the initial sum of $1,100,000 to activate the programme which was recommended in this report. This amount was allocated for basic research as well as for teaching.[2]

The Mayo Clinic has extended the scope of physical medicine so that some of the physicians taking the regular fellowship in physical medicine spend a certain time in the Institute of Technology studying biophysics, electronics and instrumentation as applied to medicine. If a physical research centre is set up at Canberra, it is to be hoped that the scope for training in physical medicine will be extended in a similar manner.

Krusen[1] has made the following statement:

> As it is now developing physical medicine is progressing into three major fields—(i) physical therapy and the employment of physical agents in diagnosis, (ii) occupational therapy, and (iii) the reconditioning of the convalescent patient.

The supplement to the Emergency Medical Services Instructions, Part 1,[3] contains the following statement:

> There would appear to be some confusion as to the meaning of the terms "rehabilitation" and "occupational therapy". The scope of rehabilitation comprises physiotherapy, occupational therapy and remedial gymnastics and games.

### Training of Undergraduate Medical Students in Physical Medicine.

The clinical teaching of physical medicine must be based on a sound knowledge of the basic sciences and on sound reasoning. Too much of the teaching in the medical curriculum in all subjects is empirical. Early teaching of the basic sciences must have a bearing on and lead up to the clinical teaching. Interchange of staff, or a system by which a staff worked in more than one department, would do much to correlate teaching. The teaching of physics should commence in the schools; in the first year of medicine only that part of the subject which has a direct bearing on future clinical work should be taught.

In the Baruch report the following recommendations are made:

> More attention than at present should be paid to biophysics as applied to medicine. . . . In the colleges and universities which give pre-medical courses more attention should be paid to the teaching of physics as related to medicine. As now constituted most of the college courses in physics stress engineering physics rather than medical physics.

Biophysics should be given its right place in the teaching of physiology; a proper department of biophysics should be set up, as in the case of biochemistry, and there should be a close link between this department and the department of physics.

The teaching of anatomy is still not well balanced in its application to the training of undergraduate medical students. More importance should be given to functional and surgical anatomy and less to certain obscure histological and neurological structures which have no practical application and are learnt only to be promptly forgotten.

During the clinical years care should be taken not to overload the curriculum; as much as possible physical medicine should form an integral part of instruction in other subjects. The use of physical means in diagnosis

and treatment should be indicated in all medical and surgical teaching.

The teaching of obstetrics is not complete without reference to pre-natal and post-natal exercises.

The student should be encouraged to observe all forms of treatment given to the patients either in the ward or in the physical therapy department, and to acquire a knowledge of apparatus used in diagnosis and treatment. Active learning by the students by keen observation is of a great deal more value than much passive teaching, which is often insisted on by the teachers and demanded by the students as their right. A few didactic lectures and demonstrations are necessary, but these should not be many. If the teaching of physical medicine is carefully integrated with that of other subjects throughout the medical course, four to six two-hour lecture-demonstrations will give to the students a working knowledge of the practical application of biophysics which he has studied in the preclinical period.

There are certain essentials that must exist in a clinical school before efficient training can be carried out.

1. A properly equipped physical department must be available where all forms of physical therapy can be given; it would also be advisable that some arrangements for research should be made within the department.

2. The department must be under the control of a director who has a special knowledge of physical medicine and can direct and control treatment and also give instruction to students. This position would be analogous to that of the director of any other special department.

3. To integrate all teaching, those engaged in the teaching of other clinical subjects must have a good knowledge of the application of physical agencies in diagnosis and treatment. The lack of knowledge and appreciation of its value are the greatest obstacles to the efficient teaching of physical medicine.

### Training of Graduate Students in Physical Medicine.

The training of graduate students in physical medicine constitutes a problem which is not so easily solved as the training of undergraduates.

Professor L. S. P. Davidson[4] divides post-graduate training into two sections, that of general practitioners and that of specialists. With regard to the training of general practitioners, Professor Davidson makes the following statement:

> It would appear that in the future a doctor will not be allowed to undertake private practice without spending a year as a house physician and house surgeon in some accredited hospital. . . . Attendance at various special departments of the hospital including the physiotherapy department should constitute part of this course of training.

At our medical school in Queensland the student will spend his sixth year in residence at an approved hospital, and this will eliminate the compulsory year as a resident medical officer. During this year any work carried out could be considered as post-graduate training. It should be obligatory that a certain amount of time must be spent observing the work in the physiotherapy department.

With regard to the training of specialists, Professor Davidson considers that the graduate who wishes to specialize in physical medicine should spend another year in a hospital or institute which has a large, well-equipped and well-staffed physiotherapy department. During this time he should attend advanced classes in anatomy, physiology and physics. Davidson believes that this training should be basic to other specialties, such as orthopædics, and advises several years' experience before recognition as a specialist in physical medicine:

> Our objective should be that in the future, when an individual is placed in the roster of accredited specialists in physical medicine, this should indicate to all concerned that he has the necessary education in the fundamental sciences and devoted the requisite time to learning the practice of physiotherapy under skilled direction: similarly the acceptance of a rheumatologist or an orthopædic surgeon on the appropriate roster of specialists should indicate not only that the individual has acquired an adequate knowledge of the clinical diseases and disorders in which he specializes, but that he has had a training in physical therapy sufficient to enable him to appreciate its value and to prescribe it in appropriate cases for appropriate purposes in the correct way.

At the Mayo Clinic a three years' fellowship in physical medicine is offered. This fellowship sets a very high standard of training, and it might be possible for some of our medical graduates to take advantage of these fellowships. In England a part-time course of training lasting for four months was commenced in London on March 18, 1945, in order to prepare candidates for Part I of the 1946 examination for the Diploma in Physical Medicine. This course included applied anatomy, physics, applied physics and physiotherapy.

I have just received the following information from Sir Robert Stanton Woods[5] with regard to the course of study and examination for the Diploma in Physical Medicine. To qualify for examination candidates must be graduates of two years' standing and must also produce evidence of having accomplished the following essentials: (i) of having held in recognized general hospitals at least two resident appointments (one of which must have been that of general house physician) extending over not less than twelve months; and (ii) of having completed a twelve months' course of full-time study consisting of (*a*) instruction, theoretical and practical, in a recognized medical school or institution extending over not less than four months in physics in its relation to physical medicine; and (*b*) instruction, theoretical and practical, in the physical medicine department of a recognized general hospital or institution during twelve months in the clinical, pathological and therapeutic aspects of physical medicine.

The only special training school as yet is at Bristol University, in conjunction with the Royal United Hospital at Bath and the Bath-Wells Orthopædic Hospital. All teaching medical schools in London and elsewhere in the British Isles would be recognized.

Examiners are appointed by the Royal College of Physicians and the Royal College of Surgeons. For Part I, in physics there are two examiners, one a non-medical physicist, and for anatomy and physiology there are an orthopædic surgeon and an anatomist. For Part II the examiners are an orthopædic surgeon, a professor of medicine and two physical medicine specialists. The examinations consist of written papers, *viva voce* interrogations and practical tests, and cover a very wide field.[1]

In an editorial article in *The British Journal of Physical Medicine and Hygiene* of November-December, 1943, the following points are brought out: (i) In what is the specialist to be trained and subsequently examined? (ii) Where is the training to be carried out? (iii) By whom is he to be trained and examined? With regard to (i), training in physical medicine requires first a sound theoretical and practical training in physics far surpassing the curriculum of first bachelor of medicine examination, supplementary teaching in physiological responses to physical agents, together with clinical experience of the action of these agents in states of disease. With regard to (ii), training would be carried out in large university centres or spa hospitals. With regard to (iii), teaching and examination require a body of trained specialists who can teach and examine with authority.

Until these are available, the teaching of physical medicine must be divided chiefly between a small number of experienced specialists in physical medicine whose knowledge rests largely on an empirical basis, and a larger number of senior physicians, orthopædic surgeons and scientists who may be without either special knowledge or personal experience in physical medicine.

It is time due consideration was given to a post-graduate course leading to a diploma in physical medicine here in

[1] As appendices I attach the following: (i) the syllabus for the Diploma in Physical Medicine of the Royal College of Physicians of London and of the Royal College of Surgeons of England, (ii) the syllabus of the Mayo Clinic, (iii) the syllabus for undergraduate teaching in physical medicine to medical students at the University of Queensland.

Australia. *The Medical Acts*, 1939, of Queensland make provision for registration as a specialist in physical medicine, but no arrangements have been made for training or examination. The scheme as suggested by Professor L. S. P. Davidson, of Edinburgh, would appear to be suitable. In such a scheme the basic studies would be common to many specialties. Such a course of studies might well be started in the university of each of the Australian capital cities or at a centre such as Canberra, where it is anticipated that advanced physical studies will be available.

The problems with us are the same as those mentioned in England, most important of which is the dearth of well-trained specialists who can teach and examine with authority. There is also a great need to improve the facilities for training in physical medicine.

### Training of Physiotherapists, Occupational Therapists and Physical Educationists.

There is no need to go closely into the syllabus of training of physical therapists, occupational therapists and physical educationists. It is essential that these courses should be given in association with a university and that they should be of a high standard. A certain amount of uniformity throughout Australia should be the aim. These forms of therapy are closely linked up with rehabilitation, so that each branch should have a knowledge and appreciation of the other if correct and evenly balanced treatment is to be given.

In Queensland we have a diploma course within the Faculty of Medicine. I am anxious that a degree should be granted especially for those who would wish to qualify for teaching or other special work. This degree course could replace the diploma. It could be a separate course of studies, still retaining the diploma; or it could be built up on the diploma by strengthening the first year subjects and the giving of an extra year of special work and study.

At the University of Queensland, as a prerequisite for the Diploma in Physiotherapy, senior physics and three other senior subjects, including English, or four senior subjects and physics to junior standard, will be necessary from November, 1948.

### Summary.

1. An attempt is made to define physical medicine.

2. The scope and importance of this work are discussed.

3. The training of undergraduate medical students in physical medicine is considered from the viewpoints of preclinical teaching, including instruction in physics, relevant anatomy and physiology, and of clinical teaching, which is subdivided into two sections, specialized teaching and teaching which is included in the instruction in medicine, surgery, obstetrics and specialties.

4. Post-graduate training is divided into training which is necessary for the general practitioner and that which is necessary for specialists in this and allied subjects. Stress is laid on the necessity for proper teaching facilities, most important of which is the well-equipped physical department, and on the necessity for qualified teachers.

5. The possibility of commencing in Australia a post-graduate course of instruction for specialists in physical medicine is considered.

6. The training of physiotherapists, occupational therapists and physical educationists is discussed. The teaching should be carried out within a university; it must be of a high standard and eventually lead to a degree course. The granting of a diploma after three years' work is desirable.

### References.

(1) F. H. Krusen: "The Expanding Field of Physical Medicine", *Proceedings of the Staff Meetings of the Mayo Clinic*, December 26, 1945, page 497.
(2) "Report of the Baruch Committee on Physical Medicine", April, 1943.
(3) "The Provision for Rehabilitation", Medical Services Instructions, Supplement to Surgery, Supplement 28A.
(4) L. S. T. Davidson: "The Teaching of Physical Medicine", *The British Journal of Physical Medicine and Industrial Hygiene*, July-August, 1944, page 78.
(5) R. Stanton Woods: Personal communication.

### Appendix I.

*Syllabus for the Obtaining of the Diploma in Physical Medicine (D. Phys. Med., R.C.P. Lond. and R.C.S. Eng.).*

This syllabus is for the guidance of candidates, and must not be considered to be complete. Attention will be directed to principles rather than to details.

Part I. (*a*) *Anatomy*:

Anatomy of the central and peripheral nervous systems.
Anatomy, mechanics, and innervation of muscle movement.
Surface anatomy.

(*b*) *Physiology.*

Physiology of movement.
Physiological response to exercise.
Fatigue.
Principles underlying nutrition—growth and repair of tissues.
Physiology of muscle and nerve; including normal and abnormal responses to electrical stimulation.
Biological effects of electrical currents of various kinds—e.g. Faradic, Continuous, High Frequency.
Electrical conduction in tissues.
Production of electrical changes in the body.
Effects of variation of atmospheric pressure, humidity, light and temperature on the body.
Principles of ventilation.

(*c*) *Physics.*

Laws and principles of electricity, magnetism, light and heat, with special reference to their clinical application.
Candidates will be required to show a knowledge of the apparatus involved.

Part II. *Clinical, Pathological and Therapeutic.*

(1) Diagnosis and pathology of diseases in which special physical methods are employed as an adjunct to diagnosis or treatment.
(2) Diagnosis of diseases and injuries of the locomotor and nervous systems.
(3) Pathological conditions in muscle and nerve.

*Therapeutics.*

(*a*) Preventive: methods of maintaining and improving physical fitness.
(*b*) Remedial: Massage and remedial exercises. Occupational therapy. Methods employed in rehabilitation centres. Electrotherapy. Actinotherapy. Hydrology and climatology. Radiant heat. Therapeutic pyrexia.

Candidates will be required to show a knowledge of the apparatus involved.

[COPY.]
From Sir Robert Stanton Woods,
London Hospital, E.1,
Bishopsgate 9284,
November 11, 1946.

D. Phys. Med. in the Royal Colleges.

Supplementary information as promised.

Physiology and Anatomy—
Viva. 15 minutes. Both examiners.

Physics—
3 hours. Bench work. Two examiners.
20 minutes—viva and apparatus—limited to wiring and the principles underlying the functioning of the apparatus as distinct from the actual theoretical or even the pathological effects on tissue. Candidates often examined on the type of fields between short-wave electrodes or with the cable electrodes.

### Appendix II.

*Syllabus of the Mayo Clinic.*

I. *Training in certain fundamental subjects.*

A. Physics of light, heat, electricity and mechanics, as applicable to physical medicine.

B. Anatomy: Special study of the anatomy of the peripheral nervous system, the muscles, blood vessels, bones and joints, special attention being paid to kinesiology and topographic anatomy; also a study of pathologic anatomy, with particular reference to trauma.

C. Biophysics with special reference to its relation to physical medicine.

D. Physiology of muscle, nerve, blood, circulation and digestion, with particular reference to the application of physical agents for the correction of abnormal physiologic processes (the physiologic basis of physical medicine).

II. *Training in research.*

A. At least one assigned or selected problem in laboratory research on some subject related to physical medicine. Special research in some such subject as: (1) Biophysics in relation to physical medicine. (2) The effects of physical agents on local physiologic processes. (3) Problems in the physiologic effects of local or general applications of heat, and (4) studies in bodily mechanics and so forth.

B. At least one assigned or selected problem in clinical research on some phase of the clinical application of physical agents. Special clinical research in some such problem as: (1) a comparative study of the surface temperatures produced by the application of various physical heating agents, (2) a controlled clinical study of the effects of local heat in chronic or acute inflammatory processes, (3) a study on the effect of corrective exercises in the treatment of scoliosis, (4) a controlled clinical study of the most effective physical measures to be used in the correction of contractures, and (5) a study on the use of hydrogymnastics in the treatment of flaccid paralysis, and so forth.

III. *Actual clinical and hospital training in the study and treatment of patients by means of various physical agents in the physical therapy department of a teaching hospital.*

A. This includes work in light therapy, thermotherapy, fever therapy, hydrotherapy, electrotherapy and mechanotherapy.

B. In addition to this work in the hospital physical therapy department there may be assignments or electives in subjects allied to physical medicine, according to the individual wishes of the student when possible, that is, orthopedics, arthritis, peripheral vascular diseases, dermatology, roentgenology, pediatrics, general medicine or surgery.

C. During this phase of clinical study the student should prepare, or collaborate in preparing, at least one scientific paper on some subject related to physical medicine in such a manner that it will leave no doubt as to his familiarity with the subject or with its presentation in proper literary style.

D. Training can be obtained in the art of medical writing, cultural studies in history, foreign languages, literary style, speaking before groups and so forth.

E. Also, there are seminars in physical medicine, and training in administration, record keeping and handling of personnel. An opportunity is offered for the development of new types of physical devices for the treatment of disease.

## Appendix III.

*Syllabus for Undergraduate Training in Physical Medicine for Medical Students in the University of Queensland.*

*Physical Medicine or Physical Therapy.*

*Definition*:

Groups.—1. Thermo-therapy.
2. Light therapy or actino-therapy.
3. Electro-therapy.
4. Mechano-therapy (massage, exercises and mechanical apparatus).
5. Hydro-therapy.

1. *Thermo-therapy.*

(*a*) *General.* Method of production; effects on circulation, other effects and bactericidal effects; technique of application; indication for application.

(*b*) *Local.* Sources, radiant heat, conversion heat and heat by conduction; physiological effects; contra-indication.

2. *Actino-therapy.*

(*a*) *Physical consideration.* Wave-length; electro-magnetic spectum; penetration of rays; sources of different rays.

(*b*) Summary of physiological and bactericidal effects produced by ultra-violet rays.

(*c*) Summary of physiological effects produced by exposure to luminous and infra-red radiation.

3. *Electro-therapy.*

(*a*) *Physical considerations.* Direct or constant current; the interrupted current; the alternating currents.

(*b*) Effects of the different types of current.

(*c*) Uses of the *different types* of current. Therapeutic; diagnostic.

(*d*) *High frequency currents.* Long and short wave diathermy.

4. *Mechano-therapy.*

(*a*) *Massage.* Definition; principles of effects; types of effects; indications; contra-indications.

(*b*) *Exercises.* Principles of muscle action; different types of movements; effect of exercise on the different systems of the body.

(*c*) *The use of remedial exercises in*—general rehabilitation; abnormal conditions of the heart; lungs; abdominal organs; nervous system; in obstetrical practice; in congenital and acquired deformities and traumatic conditions. (Hydro-therapy and mechanical apparatus are not discussed.)

*The demonstration will include*:

(1) *Thermo-therapy.* Heating by infra-red lamp; luminous and non-luminous; "bakers"; paraffin baths or packs.

(2) *Light therapy.* Ultra-violet lamp and methods of application; use of luminous heat in "baker"; luminous and non-luminous lamps.

(3) *Electro-therapy.* Galvanic or direct current; interrupted current and its effect on muscle (surging galvanic current); clonic and tonic contractions; therapeutic effects including ionisation; electro-diagnosis; diathermy (spark gap, short wave, methods of application).

(4) *Mechano-therapy.* Massage in all forms. *Movements.* Concentric and eccentric; passive relaxed; free; active (assisted, free, resisted); individual movements, group movements, co-ordinated movements; gripping reflex, postural reflexes; muscle fatigue; system of levers in muscle action; how exercises are given to increase mobility (actively rapid and in full range); how exercises are given to grade up power; how exercises are given to co-ordinate movements (contractions and relaxation, relaxation exercises).

*Remedial exercises in*: general rehabilitation; abnormal conditions of the heart; lung (injuries to chest wall, etc., asthma, other lung conditions, different types of breathing); abdominal organs; nervous system (spastic, flaccid, incoordination, tabes dorsalis, etc.); pre- and post-natal exercises; exercises for deformities of the trunk and limbs.

## Appendix IV.

*Memorandum from Dr. E. S. Meyers, Dean of the Faculty of Medicine, University of Queensland.*

Since 1942 the Faculty of Medicine of the University of Queensland has given much thought to and spent a great deal of time in devising a new medical curriculum. This new curriculum will come into force at the beginning of 1947, and embodied in it are the following provisions which have a bearing on the teaching of physical medicine.

It is stated in the matriculation requirements that no student shall be admitted to the course unless he has passed the Senior Public Examination in Physics and Chemistry.

With this background students study physics in the first year of the course with particular reference to medical application. Thus, in the first term, they revise and unify knowledge acquired during pre-university years, and in the second term the curriculum includes a study of physical phenomena important to the understanding, investigation and control of living material.

In the second and third years of the course much attention is to be given in the Department of Physiology to the study of biophysics. The Professor of Physiology (Professor D. H. K. Lee) considers that somewhere about forty per cent. of the work of his department is related to the subject of biophysics. A biophysicist is shortly to be appointed to the staff of the Department of Physiology.

Doctor Harold Crawford, who has devoted much time in Queensland to the development of physical medicine, in his capacity as Chairman of the Board of Studies in Physiotherapy, is responsible for the students' work in the fourth and succeeding years.

The course consists of a certain number of lectures and lecture-demonstrations, the demonstrations taking place in the Physiotherapy Department of the Brisbane Hospital. It is hoped that in the not too distant future a specialist in physical medicine will be appointed to the staff of the Brisbane Hospital, and that additional facilities will be provided so that students may be instructed in all practical aspects of physical medicine.

It is felt that there is a very great scope for physical medicine in the everyday practice of the doctor. One reason why much of this type of work has fallen into the hands of quacks is that arrangements in the past for the teaching of physical medicine to medical students have been most unsatisfactory.

It is hoped that before very long this criticism will not apply to medical education in Queensland.

# CROSS-INFECTION OF WOUNDS IN A SURGICAL WARD DURING A TRIAL OF THE USE OF OILED BLANKETS.

By PHYLLIS M. ROUNTREE,
*From the Fairfax Institute of Pathology, Royal Prince Alfred Hospital, Sydney.*

A PREVIOUS investigation by Rountree and Armytage (1946) revealed the large bacterial load carried by hospital blankets. It was therefore decided to conduct a trial of the use of oiled blankets in a general surgical ward in order to determine whether the use of such blankets would have any effect on the incidence of wound cross-infection.

### Plan of the Trial.

The surgical ward selected has been used in previous investigations. It contains 24 beds, 12 in the male part of the ward and 12 in the female part. These portions are separated by glass doors and a passage, from which open off the kitchen, linen store, dressing room and other small rooms. In addition there is a number of beds on a veranda, which were not included in the trial, but to which and from which patients in the main ward were transferred from time to time. The surgeon, resident medical officer and nursing staff are common to both parts of the ward. The only difference between the two parts of the ward was that the beds in the male ward were furnished with oiled blankets exclusively, while the female ward beds carried the ordinary hospital blankets and served as controls. The trial commenced on August 2 and lasted until November 30, 1946, a period of seventeen weeks.

### Treatment of Blankets.

The necessary number of blankets was collected, marked distinctively, laundered and oiled before the commencement of the trial. One hundred and eight blankets were used; this number was considered likely to be sufficient for the duration of the experiment. These were washed in the hospital laundry in the usual way in three batches of 36 blankets each, and after the final rinse were oiled with a "Fixanol C" oil and water emulsion. This emulsion consisted of the following components: water, 60 pounds, white oil, 15 pounds, "Fixanol C", 6 pounds. Twenty-four pounds of this emulsion mixed with water to give a 1 in 10 dilution were used for each load of 36 blankets. The average oil content on drying was 5% and the concentration of "Fixanol" was calculated to be approximately 1 in 800 (Rountree, 1946).

The treated blankets remained in continuous use during the four months' period of the trial. Since the number of blankets on any one bed is often altered, it was not practicable to arrange for the exclusive use of a set of blankets for each individual bed. All oiled blankets therefore came out of and were returned to the common pool as the occasion required.

At the conclusion of the trial samples were taken from unused oiled blankets stored in the linen cupboard, from oiled blankets on ten different beds in the male ward, and from unoiled blankets on ten different beds in the female ward. The unoiled blankets may or may not have been in use as long as the oiled blankets, but serve as a typical sample of the normal hospital blankets. These samples were examined for their total numbers of bacteria per gramme of blanket and for the presence of hæmolytic streptococci.

### Examination of the Flora of the Wounds.

Swabs were taken from the wounds of all patients the day before the commencement of the trial, and thereafter at intervals of one week during the course of the test. In a few cases more than a week elapsed between operation and the first removal of the dressing. In such cases the wound was swabbed when the dressing was removed. The swabs were plated on blood agar and incubated aerobically. Organisms isolated were identified as completely as possible. Hæmolytic streptococci were grouped but not typed. All staphylococci were examined for coagulase production and, as far as possible with the range of phages available, for phage type according to the method of Wilson and Atkinson (1945).

### Examination of Ward Dust and Air.

During the trial the ward dust was examined for the presence of hæmolytic streptococci and coagulase-positive staphylococci. Bacterial counts were also taken of the air in the male ward during bed-making and sweeping.

### Effect of Oiling on the Bacterial Load of the Blankets.

In a previous communication (Rountree and Armytage, 1946) a method of determining the bacterial load of blankets by the use of an air pump was described. However, this method is not applicable to the sampling of oiled blankets, as they release practically no fibre or organisms when suction is applied. Another method of sampling was therefore employed in the present investigation. A piece of blanket about three inches square was cut from the top centre edge of each blanket. One gramme of this was weighed and cut up into 30 or 40 small pieces, which were placed in 99 millilitres of quarter-strength Ringer's solution. Each sample was shaken 200 times and then the appropriate dilutions and plates were made in the usual manner. Both oiled and unoiled blankets were treated in the same way, so that comparable counts might be obtained. With plates poured from one-millimetre aliquots of the 1 in 100 dilution of the oiled blankets, the concentration of "Fixanol" in the plates is less than one part per million, which is well beyond the bacteriostatic value of this antiseptic for any organisms likely to be present in the blanket material. The counts obtained with three unused oiled blankets were 1,000, 2,000 and 2,200 organisms per gramme of material. The counts obtained with nine oiled blankets which had been in use for four months are shown in Table I, together

TABLE I.
*Bacterial Numbers per Gramme of Blanket.*

| Oiled. | Unoiled. |
|---|---|
| 81,000 | 34,000,000 |
| 500,000 | 100,000 |
| 350,000 | 22,000 |
| 25,000 | 1,800,000 |
| 18,000 | 300,000 |
| 21,000 | 980,000 |
| 30,000 | 240,000 |
| 600,000 | 6,700,000 |
| 18,000 | 2,100,000 |
| — | 16,000,000 |
| Total 1,643,000 | 62,242,000 |
| Mean 180,000 | 6,200,000 |

with the counts from ten blankets in the control ward. It will be seen that oiling and antiseptic treatment have reduced the mean count from 6,200,000 organisms per gramme of untreated blanket to 180,000 per gramme, a reduction of 97%. These figures, particularly those from the ordinary blankets, also illustrate the great variability in the bacterial load of individual blankets, ranging from 22,000 per gramme in an apparently new blanket to 34,000,000 per gramme in one which showed no obvious signs of "dirtiness". The tenth blanket sample taken in the male ward proved on examination to have come from an untreated blanket which had been used in this ward by error. It gave a count of 8,500,000 organisms per gramme, which can be compared with the mean value obtained from the other oiled blankets examined from the male ward. No hæmolytic streptococci were isolated from any of the blankets.

Air samples taken in the male ward during bed-making supplied confirmatory evidence of the value of the oiling

process in reducing the bacterial load in the blankets and so indirectly in the air. A count of 134 organisms per cubic foot of air was obtained under conditions similar to those which had previously given a count of 636; however, as soon as sweeping commenced in the ward the count rose to 634 organisms per cubic foot of air.

### Incidence of Wound Infection.

The wounds examined included all types of general surgical wounds. The majority were in such positions that they could be considered "clean" surgical wounds; but a number of cases included such conditions as chronic empyema sinuses, varicose ulcer, third degree burns, excisions of *fistula in ano* and pilonidal sinus. Such wounds were classified as "dirty" wounds. In the cases of "clean" wounds, cross-infection was considered to have occurred if the first swab, taken a week after operation, yielded a culture of microorganisms, if a culture followed a previously unsuccessful attempt at obtaining a culture, or if another type of organism appeared in a wound already yielding bacteria. In the case of the "dirty" wounds, the criterion of cross-infection was the appearance in the wounds of another type of organism. In three cases infection was apparent at the first removal of the dressing after operation, and may reasonably be assumed to have occurred in the operating theatre. In the remaining cases the infections are assumed to have taken place in the ward, and in many of these there is conclusive evidence that this was what had actually occurred.

A total of 82 wounds was examined, 43 being from the male part of the ward and 39 from the female part. In Table II is set out the distribution of "clean" wounds, "clean" wounds which became infected, "dirty" wounds and "dirty" cross-infected wounds.

Table II.
*Incidence of Wound Infection in a General Surgical Ward.*

| Wounds. | Number of Male Patients. | Number of Female Patients. |
|---|---|---|
| "Clean" .. .. .. | 17 | 16 |
| "Clean", cross-infected .. | 17 | 14 |
| "Dirty" .. .. .. | 7 | 5 |
| "Dirty", cross-infected .. | 2 | 4 |
| Total .. .. | 43 | 39 |

It will be seen that in the male ward cross-infection occurred in 44·1% of wounds and that in the female ward the rate was 46·2%. There is no statistically significant difference in these rates.

It should be noted that the criterion of infection was bacteriological and not clinical. Table III analyses in detail the types of bacteria recovered from these cross-infected wounds.

The most frequently occurring organism was the staphylococcus, and if coagulase production is taken as a criterion of pathogenicity, pathogenic staphylococci were recovered from 18 wounds in the series. There were only three cases of infection with group A hæmolytic streptococci. In general the other organisms isolated cannot be considered wound pathogens; but their isolation from surgical wounds can be taken as an index of the presence of conditions which permit the transference to such wounds of bacteria, including potential pathogens.

### Source of Staphylococcal Wound Infection.

Wilson and Atkinson's work (1945) in developing a series of staphylococcal bacteriophages, by means of which at least 23 different phage types of staphylococci may be recognized, has already proved of considerable value in epidemiological investigations. Their series of staphylococcal type cultures and a number of their phages were available for use in this investigation. Owing to difficulties in the transport from England of the phage filtrates, the complete range of filtrates was not available; but there were sufficient for the identification of 15 types. Two additional phages isolated in this laboratory and differing from the types defined by Wilson and Atkinson were also used. Wilson and Atkinson state that they were able to identify 60% of the staphylococci which they examined by this method.

The 18 strains of staphylococci isolated from the cross-infected wounds were examined for their phage type, and Table IV sets out the distribution of phage types among these strains.

Table III.
*Incidence of Various Bacteria Recovered from Cross-Infected Wounds.*

| Organism. | Number of Wounds Yielding the Organism. | |
|---|---|---|
| | Male Ward. | Female Ward. |
| Hæmolytic streptococci, group A .. .. .. | 3 | 0 |
| Coagulase-positive staphylococci .. .. .. | 9 | 9 |
| Coagulase-negative staphylococci .. .. .. | 6 | 8 |
| *Bacterium coli* .. .. .. | 2 | 1 |
| Diphtheroid bacilli .. .. | 0 | 3 |
| *Bacterium aerogenes* .. .. | 1 | 1 |
| *Streptococcus viridans* .. .. | 0 | 1 |
| *Bacillus alkaligenes* .. .. | 0 | 2 |

Four strains were not acted on by any of the phages available. It will be noted that two strains are included in Table IV as a sub-type of type 2. These two strains gave identical reactions with some of the type 2 phages, but do not fall into any of the sub-types of type 2 as defined by Wilson and Atkinson. The most interesting feature of the distribution of the phage types is the fact that nine out of the eighteen strains isolated belong to the same type—type 2B. There was also a further wound infection with this type which is not included in the table, in a patient on the veranda. This strain was first isolated at the beginning of the investigation from an infected thyreoidectomy wound. It appeared in September in two more wounds, in October it was found in five

Table IV.
*Distribution of Staphylococcal Phage Types.*

| Phage Type. | Number of Strains. |
|---|---|
| 1B .. .. .. .. | 1 |
| 2B .. .. .. .. .. | 9 |
| 2D .. .. .. .. .. | 1 |
| Subtype of 2 .. .. .. | 2 |
| 5 .. .. .. .. | 1 |
| Not typable .. .. .. | 4 |
| Total .. .. .. | 18 |

wounds, and in November in two. Evidence that cross-infection with this type had actually occurred in the ward and not at operation is available in a number of cases, in which it appeared following negative swab findings or in which it replaced a different phage type of staphylococcus. Two cases illustrating this may be cited.

One patient, R.M., had a chronic empyema sinus. On August 1, 1946, this sinus contained an untypable *Staphylococcus aureus* which persisted in the thoracoplasty wound following operation on August 13 until October 3, when no culture was obtainable. When the wound was nearly healed it broke down again, and on October 24 was found to contain the type 2B organism, which persisted for another month.

A similar course of events occurred in another case, that of E.L., who had a chronic empyema sinus. In this case the type 2B organism appeared on October 10, 1946,

replacing a type 15 strain, which had been present in the sinus before operation, and whose origin was probably endogenous, since this patient also carried type 15 staphylococci in her nose.

In October, when it became apparent from the occurrence of eight cases of type 2B infection in both male and female patients that this organism might be regarded as the cause of a large proportion of the staphylococcal cross-infection in the ward, a search was made for the source of the organism. Ten dust samples were taken from the male and female wards and from the room where the sterile dressing trays are prepared. A considerable number of coagulase-positive staphylococci was isolated from the ward dust, but none from the dressing room. Examination of 12 representative colonies so isolated showed that none of these belonged to phage type 2B, nor were they types which were present in any wounds. In one instance the type that was present in one patient's nose was found in the dust underneath that patient's bed. At the time when the dust samples were taken, six patients in the ward had wounds infected with type 2B staphylococci, and three of these were also nasal carriers of this type. Its non-recovery from the dust was therefore unexpected and may have been due to sampling technique. However, it was isolated from an air sample taken in the male ward at the end of November during bed-making and sweeping, at a time when one patient in the ward had a pyelolithotomy wound infected with it.

Nose and throat swabs were taken from the nursing staff of the ward on October 24 and 25. Coagulase-positive staphylococci were isolated from five of fourteen nose swabs and from one of fourteen throat swabs. These were typed and it was found that two of the nurses were nasal carriers of the type 2B organism. The noses of these nurses were swabbed at weekly intervals in the following weeks and they were found to be persistent nasal carriers of this type. A point which seems of considerable importance in elucidating the origin of the cases of wound cross-infection with this type is the fact that these two nurses were senior nurses who were responsible for the dressing of the wounds.

The fact that three of the patients infected with this organism were also found to be nasal carriers may possibly indicate that the infection in these cases was autogenous. On the other hand, other infected patients did not carry the organism in their noses and it seems more likely that the nasal strains were implanted during the patients' stay in the ward. A further point which supports this presumption is the fact that all type 2B strains isolated in the ward were penicillin-resistant. A survey made by Anderson (quoted by North *et alii*, 1946) indicates that penicillin-resistant staphylococci are rare in the normal population.

This phage type has been isolated from two other nasal carriers in the hospital. One of these strains, also from a senior nurse, was penicillin-resistant, the other was penicillin-sensitive. Until a large-scale survey of the phage type distribution and penicillin sensitivity of nasal staphylococci in the hospital staff is carried out, it will not be possible to say whether this type is widespread or not.

### Discussion.

The efficacy of the "Fixanol" and oil treatment in reducing the bacterial load of blankets is illustrated by the bacterial counts obtained with the blankets used in this investigation. In a report by the Commissions on Acute Respiratory Diseases and on Air-Borne Infections (1946) it is stated that oiling of blankets reduced the numbers of bacteria released by 90% to 95%. It would appear that treatment with "Fixanol" and oil not only reduced the numbers of bacteria releasable from the blankets, but also resulted in the actual destruction of a large proportion of the microorganisms deposited on the blankets in the secretions of the patient.

However, it is apparent from the incidence of wound cross-infection during the trial that oiled blankets used alone and without other dust-control measures had no effect on the control of cross-infection under the conditions obtaining in the ward during the investigation.

The relative importance of the various factors concerned in the production of wound infection in the ward is not easy to assess. With regard to the three wounds infected with hæmolytic streptococci, no information is available as to their origin. These infections occurred sporadically and there is no obvious connexion between them. Further, no hæmolytic streptococci were isolated from any of the blankets examined or from the ward dust. It is apparent that, during the period of the trial, pathogenic staphylococci were a much more frequent cause of surgical wound infection than streptococci. The impression has grown during recent years that, in this hospital, staphylococci are occurring more frequently than hæmolytic streptococci in surgical wounds, and these observations confirm this impression.

In the 10 cases of infection with type 2B staphylococci the weight of the evidence suggests that the nurses doing the dressings were responsible for some at least of the infections. It is not possible to say where or when they acquired their nasal organisms. Although they were masked while doing the dressings and the "no-touch" technique was used, it is highly probable that they were contaminating their environment. Recent observations by Hamburger and Green (1946) have shown that nasal carriers of hæmolytic streptococci release large numbers of organisms onto handkerchiefs and hands during nose-blowing. Similar observations have been made by Hare and Mackenzie (1946), using cultures of *Bacillus prodigiosus* instilled into the nose. It is not unreasonable to assume that the same contamination occurs with nasal carriers of staphylococci. Williams (1946) has demonstrated the high frequency of correlation between nasal and skin carriage of *Staphylococcus aureus* of the same phage type. During the movements incidental to wound dressing the release of staphylococci from the previously infected clothing of the nurses could readily occur.

The staphylococcal strains found in the ward dust were not responsible for any of the infected wounds. However, the type 2B organism was isolated from the air on one occasion during bed-making and sweeping, and this suggests that, if a more thorough search had been made, this organism might have been found more frequently.

The origin of the staphylococci other than the type 2B strains has not been determined. They may have come either from other patients via the ward air or dust or from the patients themselves, since it was not found possible to examine nose and throat swabs of all patients as a routine measure.

The occurrence of a penicillin-resistant staphylococcus as the chief infecting organism in this investigation raises a further point of interest. In a report on a study of the occurrence of penicillin-resistant staphylococci in infected war wounds, North, Christie and Rank (1946) state that they found that the bulk of the penicillinase-producing staphylococci which they isolated occurred only in patients during or after intense penicillin therapy. A large proportion of these strains was of the same serological type. They suggest that a possible explanation of this lies in the stimulation by penicillin which changes potential penicillinase-producing staphylococci into actual penicillinase-producing variants. They consider that ward cross-infection played little part in their series of cases. In the cases reported in this paper it is apparent that the occurrence of penicillin-resistant staphylococci in the wounds was clearly due to cross-infection in the ward. The nasal carriers and the cases of wound infection occurred without any relation to the use of penicillin therapy.

To sum up, this investigation has once more emphasized the complexity of the problem of cross-infection of surgical wounds. The evidence available suggests that in this trial infective particles from the blankets played little part in the production of cross-infection. The role of dust infected from other sources—for example, from the bed linen, or directly from the skin and naso-pharynx of patients—is difficult to assess. It appears that some of the nursing staff, directly or indirectly, may have been responsible for a proportion of the staphylococcal infections.

**Summary.**

1. Blankets treated with oil and "Fixanol" and in constant use in a surgical ward during a period of four months had a bacterial content 97% less than ordinary hospital blankets in similar use.

2. The use of oiled blankets had no effect on the incidence of cross-infection in a series of general surgical wounds.

3. Of 82 surgical wounds, 18 were cross-infected with pathogenic staphylococci and three were infected with group A hæmolytic streptococci. The total number of cross-infections in the wounds was 37.

4. Half the infection with pathogenic staphylococci was due to a penicillin-resistant phage type 2B *Staphylococcus aureus*.

5. Two nurses who did the dressings in the ward were found to be persistent nasal carriers of this organism.

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**Bibliography.**

Commission on Acute Respiratory Diseases and the Commission on Air-Borne Infections: "A Study of the Effect of Oiled Floors and Bedding on the Incidence of Respiratory Disease in New Recruits", *The American Journal of Hygiene*, Volume XLIII, 1946, page 120.

M. Hamburger, junior, and M. J. Green: "The Problem of the Dangerous Carrier of Hæmolytic Streptococci. IV. Observations upon the Role of the Hands, of Blowing the Nose, of Sneezing and of Coughing in the Dispersal of these Micro-organisms", *The Journal of Infectious Diseases*, Volume LXXIX, 1946, page 33.

R. Hare and D. M. Mackenzie: "The Source and Transmission of Nasopharyngeal Infections due to Certain Bacteria and Viruses", *British Medical Journal*, June 8, 1946, page 865.

E. A. North, R. Christie and B. K. Rank: "Penicillin-Resistant Staphylococci in Wounds", THE MEDICAL JOURNAL OF AUSTRALIA, July 13, 1946, page 43.

P. M. Rountree and J. E. Armytage: "Hospital Blankets as a Source of Infection", THE MEDICAL JOURNAL OF AUSTRALIA, April 13, 1946, page 503.

P. M. Rountree: "The Treatment of Hospital Blankets with Oil Emulsions and the Bactericidal Action of 'Fixanol C' (Cetyl Pyridinium Bromide)", THE MEDICAL JOURNAL OF AUSTRALIA, April 20, 1946, page 539.

R. E. O. Williams: "Skin and Nose Carriage of Bacteriophage Types of Staph. Aureus", *The Journal of Pathology and Bacteriology*, Volume LVIII, 1946, page 259.

G. S. Wilson and J. D. Atkinson: "Typing of Staphylococci by the Bacteriophage Method", *The Lancet*, May 26, 1945, page 647.

## NUTRITIONAL DEFICIENCY DISEASES.[1]

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IT is a difficult task to discuss nutritional deficiency diseases at a meeting of this type in the allotted time. I propose, therefore, to limit my remarks mostly to the influence of deficiency disorders on the central nervous system and its appendages, these disorders being the common bond of interest between our two sections, with brief reference here and there to other matters.

In the Goulstonian Lectures of 1938, Ungley approached his subject, "The Origin and Detection of Nutritional Deficiencies", in the following manner:

> I shall consider causes under the following headings:
> Factors adversely affecting food intake;
> Factors leading to impairment of digestion and absorption;
> Deficiencies of essential food elements conditioned by the composition of the rest of the diet;
> Factors increasing the demands for nutrient elements or the rate at which they are katabolized; and other factors.

These headings embrace a wide field, but if you will bear in mind those words, not as subdivisions of a lecture, but as advice in the manner of your approach to the subject, you will find them words of wisdom. In any circumstances of nutritional deficiency, obvious or suspect, or indeed often in the circumstance of a difficult case this advice is useful.

Nutritional deficiency embraces deficiency of caloric requirements, deficiency of variety of foods, especially protein and carbohydrate, fluid deficiency, vitamin deficiency, mineral deficiency, oxygen deficiency, and infinite admixture of these; indeed, one can say dogmatically that in clinical medicine a pure single substance deficiency disease is never encountered.

I propose further to limit my remarks mainly to vitamin deficiencies which may be of common interest, and to protein deficiency.

### Fat-Soluble Vitamins.

#### *Vitamin A.*

The most clear-cut effect of vitamin *A* deficiency is night blindness. Probably liver was used in the treatment of night blindness about 1500 B.C. Early in the twentieth century the influence of a substance contained in fat on various ocular troubles was discovered, and knowledge has advanced steadily since then.

Vitamin *A* is obtained from the food we eat, directly from animal tissues, or indirectly from carotene, a widely scattered plant pigment, which after ingestion is converted by a liver enzyme (carotenase) to vitamin *A*. Vitamin *A*, whatever its origin, is mainly stored in the liver. Bile is an important factor of the absorption of vitamin *A* as such, and of carotene, and it is an insufficiently recognized point that paraffin and similar aperients seriously interfere with the absorption of both.

"The specific tissue changes due to deprivation of vitamin *A* is replacement of various epithelia by stratified squamous keratinising epithelium." This was the generalization of Wolbach and Howe in 1925. Bignell and Prescott add: "If this statement is broadened to include all tissues of epithelial origin be they ectoderm or entoderm . . . a valuable generalisation is made which harmonises all the more important functions of vitamin *A*, always remembering that some tissues are damaged earlier than others by deficiency."

Time does not permit the application of this generalization to all tissues.

The ocular effects which may result from vitamin *A* deficiency are xerophthalmia, Bitot's spots, asthenopia, and, of course, night blindness.

The effects of vitamin *A* deficiency on the central nervous system are less clear. Irving and Richards have produced certain changes in the central nervous system of vitamin *A* deficient rats with such constancy that they have suggested this method as a basis of vitamin *A* assay. These changes, however, have never been recognized in man.

The relationship between vitamin *A* and lathyrism has been a matter of speculation. Especially in those areas in Asia, in Egypt and so on, where lathyrism occurs, a variety of gross nutritional deficiencies is apt to obscure the position, and a number of disorders have incorrectly been described under this name. The common factors associated with lathyrism are famine and some form of paralysis of the legs, the paralysis including the distribution of the pyramidal tract; vitamin *A* lack is commonly believed to play a part, and generally there is a history that the sufferer has eaten one or other variety of vetch (*Lathyrus*), from which the name is derived.

The accepted explanation of lathyrism is that the clinical syndrome is due to poor diet and lack of vitamin *A*, the two together paving the way for a toxic agent to attack the central nervous system; such toxic agents, which may include a vetch, are not always the same and do not always produce the same symptom complex.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on November 1, 1946.

Incidentally, Mellanby considered vitamin *A* lack to be an essential factor in the incidence of toxigenic convulsive ergotism.

You may wonder why I mention lathyrism, which most of you will never see; but it seems to me that, as we become more aware of the possibility of solving some of our obscure neurological problems in deficiency disease, the basic suggestions implied in this explanation of lathyrism may have a wider application.

I feel that I may well make some passing reference to vitamin *A* and the respiratory tract, in view of the popular use of vitamin *A* preparations as a panacea for respiratory tract infections. It is not so long ago since vitamin *A* was hailed as the "anti-infective vitamin". There is no evidence that it is "anti-infective". It has its specific effect on epithelia, and there is good evidence that healthy epithelium is more resistant to infection. On these grounds its use in selected circumstances is justifiable. Vitamin *A* certainly plays an important part in the development of teeth.

### *Vitamin D.*

There are believed to be ten *D* vitamins, but so far only vitamin $D_2$ (calciferol) and vitamin $D_3$ are of any importance whatever. Calciferol, which was earlier regarded as the only vitamin *D*, probably never occurs naturally in the diet. The confusion in this regard arose when a sterol was found in animal fats, which could be irradiated to active calciferol, and which gave the absorption spectrum of ergosterol. It was therefore thought to be this substance. Ergosterol is found only in plants, especially the lower plants, and fungi such as ergot, from which it derived its name.

The sterol found in animal fats is now known to be 7-dehydrocholesterol. This is converted by sun irradiation to vitamin $D_3$ and it is probably the only important source of vitamin *D*. Thus animals gain vitamin *D* by eating the vitamin *D* containing tissues of other animals or by the direct action of the sun's rays on the pro-vitamin in or on their own skin. It is widely stored in tissues.

> Vitamin D controls a fundamental stage in the metabolism of phosphorus and calcium, being responsible for their effective absorption from the bowel and controlling their fæcal excretion.

Deficiency of vitamin *D* leads to defective absorption and excessive excretion of phosphorus and calcium. The blood levels of phosphorus or calcium or both fall, increased irritability occurs at neuro-muscular junctions, producing tetany and spasmophilia, and cessation or retardation of ossification in growing bones produces the clinical picture of rickets.

Tetany and spasmophilia are the only known effects of lack of vitamin *D* on the nervous system. There is no call to discuss this further, but I think we should remind ourselves of the danger that lurks in the injudicious use of large doses of vitamin *D*. It is the only vitamin with regard to which so far hypervitaminosis is known to exist as a matter of any significant importance. The literature now contains many references to the widespread deposition of calcium in many tissues, including vital tissues, which results from its excessive use.

## Vitamin B Complex.

The happier days when we studied only vitamin *B* are long past. We are now confronted by a whole series of vitamins in the *B* group, and if we have added to our knowledge, we must still confess that a great deal of confusion and uncertainty remains with regard to the physiology and functions of these substances.

### *Vitamin $B_1$.*

The thermolabile member of the group is probably the one of most practical interest to us in this country. Apart from other considerations, it is thermolabile and is vulnerable to bad cooking and bad handling. The League of Nations Committee many years ago estimated the minimum requirements of vitamin $B_1$ for adults at about 30 international units (one milligramme) of thiamin per day. Further investigations have suggested the desirable intake to be 600 international units. A wide survey in England at the beginning of this decade showed that only upper class diets contained as much as 500 international units, whilst working class diets were dangerously close to the beriberi level. Perhaps with the rising cost of meat, fresh fruit, vegetables *et cetera* our wage-earners too may approach the danger zone.

I do not propose to consider here the enormous amount of work which has and still is being done in the physiology of vitamin $B_1$; I merely remind you briefly of the basic function of thiamin in body metabolism. Thiamin combines with pyrophosphoric acid to form pyrophosphate, a cocarboxylase which acts on pyruvic acid, an intermediate product in the breakdown of glucose. Absence of the vitamin halts the breakdown at this stage, and pyruvates accumulate above the normal blood level of 4·7 milligrammes *per centum*. The degree of rise in the blood pyruvate level bears a relationship to thiamin deficiency, although perhaps this relationship is not exactly proportionate.

Neurological lesions resulting from vitamin $B_1$ deficiency are believed to be due to the inability of nervous tissue to metabolize glucose efficiently. It is interesting to recall that the oxidation of glucose can be inhibited by such chemical substances as alcohol, anæsthetics and barbiturates, and it has been amply shown that narcotics greatly increase the thiamin requirements of the body.

Laboratory animals and human beings living on a thiamin-deficient diet exhibit decreased glucose tolerance; but the relationship of the vitamin with glucose storage, blood sugar levels *et cetera* is still obscure, and equally obscure is the question whether these disturbances are directly related to disturbances of the nervous system.

The diminished ability to absorb thiamin when hydrochloric acid is absent from the stomach, the limited ability of man to store thiamin, and finally disproportionately large amounts of carbohydrates in the diet are some of the important factors which play a part in precipitating a deficiency. And I need hardly remind you that alcohol, especially if combined with insufficient diet or with a diet excessive in carbohydrate, may precipitate what we all sometimes see—alcoholic polyneuritis.

There are many who doubt that the vitamin $B_1$ deficiency alone is responsible for all the manifestations of beriberi and allied conditions, including even some of the classical types. Vitamin *B* has been called the "anti-neuritic" vitamin; it is by no means clear that it possesses any specific anti-neuritic property. The part it plays in carbohydrate metabolism is not limited to the carbohydrate metabolism of nervous tissue.

Endeavours were made to explain the relatively early appearance of nervous signs in the developing deficiency state, on the grounds that nerve is man's most specialized tissue and is more vulnerable to deficiency at an early stage and to a greater extent than other tissue. This still left it difficult to explain why individual nerves or centres or tracts might suffer more severely than others.

Heart muscle has great capacity for storage of thiamin, and "hangs on to it" longer than does any other tissue, including liver. While cardiac disturbances in beriberi are not uncommon, it may be this capacity of the heart to "hold" vitamin which renders cardiac complications on the whole less frequent and less frequently fatal than a careful consideration might suggest. So far as I am aware, nerve tissue has no capacity for storage. I do not think we can any longer regard the neurological manifestations of nutritional deficiency as due exclusively to lack of vitamin $B_1$—a belief which was widely accepted ten years or so ago.

The *B* vitamins are intimately concerned with and produce their effect as results of disturbances of tissue respiration. To quote briefly from Hadfield and Garrod:

> It is established beyond reasonable doubt that several vitamins are intimately concerned in the process of intracellular respiration by providing certain chemical groupings which are essential to intracellular oxidations and reductions. For this reason the manifestations of some vitamin deficiencies can be properly regarded as

the result of the functional failure of an essential enzyme reaction, which may be profound or even fatal, without the accompaniment of any recognizable histological change.

All living cells, including bacteria, obtain their energy from the intracellular oxidation of the end products of digestion. Oxidation may be brought about by the addition of oxygen, or by the removal of hydrogen, or by the loss of an electron. In many cellular oxidations the energy release is achieved by dehydrogenation. Dehydrogenation is carried out by the combined action of two varieties of catalysts: firstly the enzymes, which unite with the food molecule and activate it to yield up its hydrogen; and secondly coenzymes, which take up and carry hydrogen and electrons and thereby become reduced.

The *B* complex vitamins play a vital part in these systems of coenzymes and are therefore of basic importance in tissue respiration.

### *Vitamin $B_2$: Riboflavin.*

Riboflavin, first described by Warburg and Christian in 1932, is a yellow enzyme composed of protein and pigment, mutually necessary to each other, which plays a part in tissue respiration. It is very heat-stable, though unstable in alkali and in light. It is widely distributed in Nature. It is not definitely known to fulfil any single specific function in the metabolism of the central nervous system, but ophthalmologists have a practical interest in riboflavin deficiency.

With regard to its physiology, Bignell and Prescott make the following statement:

> The need of Riboflavin . . . is probably due to the fact that it is an essential constituent of the flavo-protein enzyme system. It is believed that if hæmin substances are *not* present in a tissue (e.g. the avascular cornea) the oxidation within the cell is accomplished by means of flavo-protein. The cornea, being avascular, contains no hæmin compounds, and therefore the greater part of interchange in oxygen is effected through flavo-protein. When riboflavin is deficient, the flavo-protein enzyme system is disturbed. This explains one of the most constant ocular changes noted in Riboflavin deficiency, namely, corneal vascularization. The proliferation of capillaries from the limbus into the cornea is explained as an attempt by the latter to overcome the local asphyxia by bringing available oxygen into closer proximity with its cells, which normally receive their nourishment from the limbic vessels or the aqueous humour.

Various other ocular troubles including cataract have been attributed to riboflavin deficiency, but I believe there is no sound evidence to support at least most of these contentions.

In the case of the laboratory dog, riboflavin has been described as responsible for degenerative changes in the central nervous system, which may develop rapidly. It has also been stated by Varotti and others that riboflavin is concerned in porphyrin metabolism.

I do not know of any definite evidence that riboflavin is directly concerned with the nervous system in man; but I believe that there is probably room for further research in this connexion. We may remind ourselves that there is a disturbance of porphyrin metabolism of still unknown origin described under the name "hæmatoporphyrinuric polyneuritis".

I make no attempt to go further with this vitamin except to remind you of the changes which occur in the tongue, on the lips, at the angles of the mouth and so forth when it is deficient.

### *Vitamin $B_3$ (Nicotinic Acid, Nicotinamide and Niacin).*

β-pyridine carboxylic acid attracted little attention when first isolated from yeast thirty years or so ago, and it was not until a derivative was successfully used in the treatment of canine black tongue, and thereafter applied to pellagra, that its importance was recognized in the last decade.

It is widely distributed in Nature, but especially in meat, liver, eggs, salmon and whole cereals. It is extremely stable, and like riboflavin, forms part of a complex enzyme system capable of undergoing reversible oxidation and reduction in tissues.

There is indirect evidence that both riboflavin and nicotinic acid are linked with carbohydrate metabolism. For instance, an increased intake of carbohydrate and of insulin by diabetics may be associated with the development of glossitis, cheilosis and stomatitis, which rapidly disappear with the administration of riboflavin and nicotinic acid. Further, the body levels of riboflavin and nicotinic acid may be greatly depleted in the diabetic ketotic state, and it is known that pellagrins are hypersensitive to insulin. The position is, however, still obscure. Spies believes that it is the part played by the nicotinic acid in cerebral carbohydrate metabolism that is responsible for the mental disturbances of pellagra.

The early name "pellagra-preventing factor", by which nicotinic acid was known, was dropped when it became recognized that clinical pellagra was a deficiency syndrome not limited to the lack of one substance. All competent observers are agreed on this. Spies considers three clear-cut syndromes can be recognized in clinical pellagra:

1. Nicotinic acid deficiency giving rise to dermatitis, glossitis, stomatitis, gastric, intestinal and mental symptoms, and lesions of vulva and perineum.
2. Riboflavin deficiency responsible for the cheilosis, perlèche, and seborrhœic dermatitis which surrounds the naso-labial folds and adjacent areas.
3. That a $B_1$ deficiency is responsible for the neuropathy.

Those who have had the opportunity of studying pellagra *en masse* do not appear satisfied that pellagra can be explained entirely on the basis of vitamin deficiencies and offer a variety of other additional suggestions.

I am concerned mostly at this moment with the neuropathies. We have, most of us, rarely encountered those cases of so-called peripheral neuritis of various origin which may extend so that upper neurones and even neurones of the intellect may become involved. Korsakoff's syndrome exemplifies this, and may occur in a variety of different toxic states. It was the late James Collier, I think, who laid such particular emphasis on these points, and came to refer to these disorders as polyneuritis or polyneuronitis, rather than as peripheral neuritis. On this basis it would seem a relatively simple matter to explain many of these psychoneurological disturbances of deficiency disease as due only to thiamin lack. Experienced observers, however, do not consider that all the neurological clinical pictures can be explained on these lines.

In contrast to the mainly peripheral and early appearing neuritis which dominates the picture of beriberi, the nervous symptoms of pellagra appear relatively late; they are produced by lesions in the spinal cord, consisting of areas of scattered, symmetrical demyelination, especially in the posterior columns and in the spino-cerebellar and pyramidal tracts. In most cases the afferent tracts suffer more than the efferent tracts, which indeed are often spared. In the brain, the frontal lobe is most frequently involved. It is said that the lesions bear some resemblance to those of subacute combined degeneration.

Joliffe described a syndrome which he believed to be due to nicotinic acid deficiency, and which he named nicotinic acid deficiency encephalopathy; the syndrome is characterized by clouding of consciousness, changing cogwheel rigidities and uncontrollable sucking and grasping reflexes. Oculomotor disturbances may also be present. This syndrome untreated is usually fatal; treatment with thiamin in adequate dosage did not affect the mortality rate, but Joliffe claims to have reduced the mortality rate to 32% with nicotinic acid.

This is an interesting syndrome described by Joliffe and his co-workers; but it has at least points of relationship with the Wernicke syndrome. The Wernicke syndrome is characterized by "clouding of consciousness, varying ophthalmoplegias and ataxia", and the associated morbid lesions are described as follows:

> The lesions are confined to the periventricular gray matter and are characterized by small foci of degeneration and varicose deformities of the blood vessels. There is subacute necrosis of the adjoining parenchyma, and small petechial hemorrhages are frequently, but

not always, found throughout the lesions. The areas most constantly involved are the paramedian and paraventricular nuclei of the thalamus and hypothalamus, the mammillary bodies, the periaqueductal region of the midbrain (nuclei of the third and fourth nerves), the abducens nucleus, the nucleus triangularis and nucleus Bechterew of the vestibular nerve and the dorsal nucleus of the vagus.

Joliffe and his co-workers examined twenty-seven cases of Wernicke syndrome and reported them in 1941. The conclusions were briefly as follows. (i) They believe that the syndrome originally described by Wernicke and since recognized as being initiated by a variety of causes, is probably a combination of several nutritional deficiencies and is not necessarily complete in any case. (ii) Results indicated that the ophthalmoplegias were due to thiamin deficiency, and that, to quote Joliffe's words, "the clouding of consciousness may be related to anything which interferes with proper cerebral metabolism, some of the known offenders being lack of carbohydrate, lack of oxygen, lack of thiamin or nicotinic acid or riboflavin or of all three, and possibly a lack of other substances now under investigation". (iii) Thirdly, they considered the ataxia as "difficult to evaluate" and its response to therapy at that time to be unknown.

These workers considered the ophthalmoplegia to be invariably preceded or accompanied by peripheral neuropathy, and suggested that this was in keeping with the earlier experimental work of Alexander, who stated that smaller doses of thiamin were necessary to treat the encephalopathic disorders than the peripheral neuropathy. Joliffe considered that delirium commonly preceded the development of the Wernicke syndrome, and that in patients who recovered, Korsakoff's syndrome commonly occurred and was not constantly responsive to thiamin.

In another paper Joliffe has shown that whatever other deficiencies may exist, thiamin deficiency is constant in the Wernicke syndrome, as is shown by the constant rise of the pyruvate level in the blood.

In reviewing the Wernicke syndrome, Campbell and Russell independently came to similar conclusions after investigation of twenty-one cases. They made the following statement:

> The encephalopathy occurred as a complication of chronic alcoholism, gastric carcinoma and other gastro-intestinal disorders, pernicious anæmia, vomiting of pregnancy, and a small group of heterogeneous disorders.

Campbell and Russell of course regarded the syndrome as one of multiple deficiencies.

J. D. Spillane, who investigated neurological disorders of prisoners of war in the Middle East, and later from Germany and the Far East, at a discussion held at the Royal Society of Medicine, classified the cases he had encountered. It is interesting to note from a personal discussion that Dr. W. Bye saw the same types of disorder in Malaya. I myself have seen only the late results in cases in which recovery did not occur promptly; but I notice on reference to Spillane's comment that I have seen most of these at one time or another. Spillane's statement is as follows:

> It is not possible to provide an ætiological or pathological classification at present, but we may discern at least six fairly distinctive, clinical types of case: (1) Retrobulbar neuritis. (2) Polyneuritis. (3) Ataxic paraplegia. (4) Spastic paraplegia and quadriplegia. (5) Wernicke's encephalopathy. (6) Atypical encephalopathic states.
>
> Other neurological symptoms and signs have been noted—anosmia, ageusia, trigeminal hypoæsthesia, tinnitus and nerve deafness, nystagmus, diplopia, facial paresis, laryngeal paresis, urgency of micturition and dysæsthesiæ of the lower limbs. In a few cases there has been intellectual deterioration.

Spillane then goes on to describe the more detailed examination of these conditions, which I do not think I need mention. With regard to ætiology, he makes the following statement:

> The deficiencies which these men suffered were undoubtedly multiple, but there is good evidence to suspect lack of some part of the vitamin B complex in the majority. If such is the case, it remains to be seen whether the neurological lesions are a direct result of such deficiency or whether the mechanism is more obscure. I have been impressed by the importance of dysentery and diarrhœa in this respect, and we may have to look further than the vitamins—to disturbance of carbohydrate, fat and protein metabolism and to the essential amino-acids.

In this connexion the observations of Captain Schwarr of the Dutch Army Medical Service are interesting. In 1942-1943 he treated large numbers of patients who suffered from various neurological deficiency disorders in an internment camp in Java, in the following manner. Six groups were given the ordinary camp diet, on which their trouble had already developed. As additions, one group was given 5,000 international units per day of vitamin $B_1$; another group received 20 milligrammes of riboflavin per day; a third group received 100 milligrammes of nicotinamide per day; a fourth group was given 160 milligrammes of vitamin *C*; and a fifth group received 100 grammes of beans per day as an addition to the diet. All groups were treated for a period of two to three weeks. In these five groups Schwarr reported that he had "no success", whereas in a sixth group he got "very good results" simply by the addition of three eggs per person per day to the ordinary prison diet. He spoke also of the benefit of meat, liver and fish which he had prescribed in other circumstances. I have not the exact details of these observations, but the results are consistent with results of other observers as reported by Bignell and Prescott.

### Other Considerations.

I have not the time on this occasion to discuss the other vitamins of the *B* group; but I should mention that there is no evidence so far that vitamin *E* is intimately concerned in the development of progressive muscular atrophy, as was suggested a few years ago.

These, then, are some of the problems which confront us when we consider vitamin deficiency in relation to neurological disorders. If we do not see beriberi and pellagra in this country, at least we occasionally encounter polyneuritis resulting from alcoholism, pregnancy, and lesions of the alimentary tract, *et cetera*, which are the result of nutritional deficiency. Moreover, subacute combined degeneration is not so very rare. If the genesis of this last-mentioned disease is still not completely understood, at least we know it to be a deficiency disturbance based on a gastric defect.

While we are considering this subject, it is as well to remind ourselves that in subacute combined degeneration, as in pellagra, the neurological changes are not always limited to the central nervous system, but may involve peripheral nerves also. It may be, therefore, that subacute combined degeneration also is not a single substance deficiency.

I do not think for purposes of this discussion that we need mention scurvy in detail. It is not associated with any but coincidental disorders of the nervous system; but in passing, Brain describes it as a rare cause of subdural hæmatoma.

You will gather from the foregoing comments several points:

1. There is a general realization that the deficiency of certain food substances, whether absolute or relative on account of defective absorption or other factors, is basically responsible for many disturbances of the central nervous system and its appendages.

2. Whilst such deficiencies largely concern the *B* complex vitamins, there is no finality of opinion as to which vitamins are responsible for which disturbances.

3. Most experienced observers believe that the vitamins are not the sole factors concerned.

And that brings me to my final point: what of the proteins? I quote from Samson Wright:

> Protein is an essential constituent of the diet because it is the only class of foodstuff containing nitrogen and sulphur in a form which can be assimilated by the tissue cells and used for the replacement of that lost as a result of normal cellular activities.

We appreciate that protein deprivation may produce nutritional œdema, that disturbances of the albumin-globulin ratio may do likewise, that certain amino acids are necessary to growth and even to life; but I venture to say that, in spite of these pictures of the gross effects as one might describe them, we still know little of the less obvious effects of protein deprivation. I would draw attention to the admirable comment on protein deficiency by C. G. McDonald in the Annie B. Cunning Lecture of 1944. It is evident, therefore, that while much work still needs to be done on vitamin deficiency, we are as yet only on the fringe of the understanding of protein deficiency.

# Reports of Cases.

## NOTE ON A CASE OF BILATERAL DUPUYTREN'S CONTRACTURE OCCURRING IN ASSOCIATION WITH SPONDYLITIS ANKYLOPOIETICA.

By GARTH MCQUEEN,
*Ashfield, New South Wales.*

MR. M., aged fifty-two years, is a sufferer from *spondylitis ankylopoietica* (Marie-Strümpell disease). The first episode in the development of this distressing affliction occurred at the age of twenty-one years, when, without any antecedent injury, a painful swelling of the foot and ankle of the left leg developed overnight. This was succeeded within a few days by painful swelling of the left knee. It was sixteen weeks before the patient was able to leave his bed and six months before the swelling of the knee and foot had subsided. In the sixteen weeks in bed he lost four and a half stone in weight. The only local residuum is a rheumatoid deformity of the second and third toes of the left foot.

About a year later he was unfortunate enough to suffer a severe injury of the right forearm, with, it appears, crushing of the ulnar nerve, the function of which was regained only after some two years.

At the age of thirty-five years he noticed the first symptoms of spinal affection, of stiffness on straightening up after work—"lumbago" as he himself describes it. In 1937, when he was aged forty-two years, he fell at work onto the base of his spine, and from this time onward rigidity of the spine progressed rapidly. He has done no work for ten years, and now the spinal deformity is such that he can barely see horizontally when standing.

Seven years ago, in 1939, contracture of the fascia of the left palm developed, and in 1946, some six months ago, the right palm became similarly affected. He now has a well-developed Dupuytren's contracture involving the fourth finger of each hand.

With regard to the ætiology of Dupuytren's contracture, the following appears in Boyd's "Surgical Pathology":[1]

> The etiology has long been a matter of debate. The extensive studies of Meyerding and his associates suggest that the condition is an inflammatory one involving the skin, subcutaneous tissue and interstitial connective tissue down to the tendon sheaths.

In Christopher's "Textbook of Surgery"[2] the following statement is made:

> Of many theories of cause, probably the only one of important association is heredity. . . .

In the article by C. E. Corlette[3] it is stated that "Dupuytren's contracture is due to hypertrophy of the palmar aponeurosis", and in the same article, on an earlier page, Horwitz[4] is quoted:

> ". . . the essential process in Dupuytren's contracture is benign fibroplasia of the palmar connective tissues. Inflammatory features are not constant, nor is there any evidence that the proliferative process arises from the walls of the blood-vessels.

The essence of Corlette's article[5] is the following:

> The still current view that Dupuytren's contracture is due to trauma or toxæmia is criticized. . . . The more likely alternative is that all cases of Dupuytren's contracture are familial, and would always be found so if the descent and complete family history were accurately known.

The only other commonly incriminated factor is trauma. Now Mr. M. is one of a family of thirteen. He has five brothers, aged between forty and fifty-eight years, and seven sisters, aged between thirty-six and sixty years. He sees all his brothers and sisters regularly; none of them shows any sign of the contracture. His father died at the age of seventy-nine years and his mother at sixty-eight years; his paternal grandfather died at 101 years, and the remainder of his grandparents all lived to considerable ages. In none of them did he ever hear of any palmar contracture. Mr. M. himself is sure that his palmar contracture is a novelty to the family.

Mr. M. had done no work for three years prior to the onset of the contracture of the left hand and for more than nine years prior to the onset of the contracture of the right hand. During the years before he was forced to retire he had worked hard at varied labouring tasks and had driven a team of horses for several of the latter years.

Thus we have a man in whom there has developed a bilateral Dupuytren's contracture years after he had given up work, whose family history, inadequately though it may be assembled, at any rate recently contains no other similar lesion, and in whom there is in progress a widespread affliction of a rheumatoid nature. It thus seems not illogical to suspect that in this case, and therefore perhaps in others, an inflammatory factor may be present.

### References.

(1) W. Boyd: "Surgical Pathology", Fifth Edition, 1942, page 777.
(2) F. Christopher: "A Textbook of Surgery, by American Authors", Third Edition, page 1616.
(3) C. E. Corlette: "Dupuytren's Contracture", THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, 1944, page 177.
(4) T. Horwitz: "Dupuytren's Contracture", *Archives of Surgery*, Volume XLIV, 1942, page 687; quoted by C. E. Corlette, *loco citato*, page 180.
(5) C. E. Corlette: *Loco citato*, page 182.

## REPORT OF TWO UNUSUAL CASES OF HYDATID MOLE.

By LILLIAN GOHSTAND,
*Brisbane.*

THE following cases may be considered unusual enough to merit publication. Their distinction lies only in the comparatively advanced ages of the patients, that of one of them being so advanced as almost to preclude in our minds the possibility of conception.

### Case I.

Mrs. N., aged fifty-five years and four months (verified by the Registrar-General's Department), was admitted to the Brisbane General Hospital on August 13, 1946. She had six children, the last confinement, at the age of forty-three years, having resulted in the birth of twins. Since then the menses had been regular until five months prior to her admission to hospital, when "floodings" occurred on two successive menstrual periods and were followed by a normal and then a missed menstrual period; for the month prior to her admission to hospital she had lost bright red blood daily *per vaginam*. For the past three weeks she had suffered continuously from nausea, anorexia and vomiting. Over the past eighteen months she had lost three stone in weight. No breast changes had occurred.

At the first examination the patient was seen to be a vigorous, middle-aged woman, vomiting clear bile-stained fluid. She had a diffuse colloid goitre of several years' standing. The pulse rate was 100 per minute and the

blood pressure was 180 millimetres of mercury (systolic) and 100 millimetres (diastolic). The ward test revealed no abnormality in the urine. In the abdomen a firm, rounded, tender mass the size of an eighteen weeks' pregnancy was felt rising out of the pelvis; there was a little guarding of the recti. The cervix and external os were softened and admitted one and a half fingers; the internal os was closed. The uterus was then recorded as being uniformly enlarged, tensely cystic and of the size of an eighteen weeks' pregnancy. Bright blood was found on the glove after the vaginal examination. Secretions were expressed from the breast and the areolæ looked "active".

A diagnosis of pregnancy complicated by threatened miscarriage and moderate *hyperemesis gravidarum* was made. Conservative treatment with isolation, sedation and fluids was instituted, and the symptoms abated gradually.

On further examination of the patient three days later, the cervix was less soft and irregular in consistency; an irregular, friable mass was palpated inside the internal os, which was now patent. The uterus had an irregular and rather flattened contour. These findings, together with the age of the patient and the history of loss of weight, raised the question of uterine malignant disease (possibly chorionepithelioma), and it was decided to treat the patient accordingly. Uterine curettage was performed on the following day and masses of placental tissue were obtained, in which unmistakable vesicular degeneration of the chorion was found. After curettage the uterus quickly decreased in size, toxæmic symptoms disappeared, and the patient made an uneventful recovery.

In view of the alleged frequency with which vesicular mole in elderly subjects degenerates into malignant disease, skiagrams of the chest were taken; however, these revealed no signs of metastases. A positive result to the Aschheim-Zondek test, a dilution of 1 in 50 being used, was obtained one week after curettage; no reactions with this dilution were obtained at four weeks, eight weeks and 12 weeks after the patient's discharge from hospital.

### Case II.

Mrs. A., aged forty-nine years, was admitted to the Brisbane General Hospital on April 2, 1946, with a history of a slight dark discharge from the vagina for four months. She had two children, aged respectively seven and four years. Since her last confinement her menstrual periods had been regular, occurring every twenty-one days and lasting for four to six days. The last normal period had occurred in October, 1945. From that date the menstrual discharge had been slight, dark and continuous, and there had been some recent brisk hæmorrhages of bright blood with clots. The breasts had been painful and enlarged two months previously, but had regressed since. Morning sickness, which had attended the previous pregnancies, had been prolonged over three months in the present instance, but had ceased three or four weeks prior to the patient's admission to hospital. She had noticed contractions of the uterus but no fœtal movements.

On examination the patient had an asthenic but otherwise healthy appearance. The systolic blood pressure was 190 millimetres of mercury and the diastolic pressure was 100 millimetres. The ward test revealed no abnormalities in the urine. The uterus was enlarged to the level of the umbilicus. It appeared gravid and a uterine souffle was heard. No fœtal heart sounds were heard. The cervix was very small and the external os was dilated sufficiently to admit one finger. No pulsation of uterine vessels was felt in the fornices.

A diagnosis of five months' pregnancy with a fœtus of doubtful viability was made. Skiagrams of the uterus made on the following day revealed no fœtal bones, and as an Aschheim-Zondek reaction was obtained with a dilution of 1 in 100, the diagnosis of hydatid mole was made. During the next few days the uterus increased rapidly in size to that of a thirty-two weeks' pregnancy. Four days after the patient's admission to hospital abdominal hysterectomy was performed and an excellent specimen of hydatid mole *in situ* was obtained. The patient made an uneventful recovery.

### Discussion.

According to Greenhill, hydatid mole occurs in 1 in 1,500 pregnancies. He further states that 4% of pregnancies which result in spontaneous abortions are hydatid moles and are unrecognized as such. The cases described were of particular interest because of the diagnostic problems which arose. DeLee states that hydatid moles occur more frequently in *multiparæ* aged over thirty-five years and have occurred in women aged up to fifty-three years; if fifty-three is the latest reported age, then Mrs. N. has created something of a record, not only in moles, but possibly in conception as well.

The diagnostic pointers to hydatid mole may be summarized as follows: (i) irregular vaginal bleeding, (ii) fulminating onset of toxic states, (iii) unduly great enlargement of the uterus, (iv) presence of vesicular material.

Irregular vaginal bleeding varies in nature and extent, and so has to be considered in conjunction with the whole clinical picture. In the first case, the rather scanty vaginal loss, the change in consistency of the uterus and cervix, and the irregular mass palpated after the patient's admission to hospital pointed strongly towards the diagnosis of *carcinoma uteri*, especially as the possibility of pregnancy seemed so remote. In the second case, however, in which the continuous vaginal loss of long duration was associated with rapid increase in size of the uterus, the diagnosis was fairly well apparent before verification by ancillary aids.

Greenhill and Melody both emphasize the importance of signs of toxæmia, especially nausea, and vomiting in the diagnosis of hydatid mole and even state that toxæmia without mole rarely occurs in the early months of pregnancy. There appears to be no correlation in these cases between the degrees of toxæmia and the size of the mole. Toxæmia was pronounced, and in fact it was the presenting symptom in the older woman, whereas it assumed a minor role in the case of the younger. Hypertension (another toxæmic sign) was present in both cases; but examination of the urine revealed no evidence of renal damage.

Rapid uterine enlargement was very obvious in the second case.

Typical vesicular material was obtained in both cases.

#### *Treatment.*

The ideal treatment (Greenhill) is thorough curettage followed by the frequent performance of the Aschheim-Zondek test with quantitative estimations of the gonadotrophin in the urine. However, in the case of women aged over forty years, because of the reputedly greater incidence of malignant degeneration, hysterectomy is sometimes desirable, especially in the presence of great enlargement of the uterus.

#### *Incidence.*

Although it is often stated to be higher, recent estimation of the true incidence of chorionepithelioma following hydatid mole gives it as not more than 2%. Dr. C. A. Thelander, under whose care these patients were admitted to hospital, in over thirty years of practice has not encountered one case of hydatid mole which was followed by the development of chorionepithelioma; further, in the few cases of chorionepithelioma which he has encountered, in no instance was there any evidence of antecedent hydatid mole.

### Acknowledgements.

I am greatly indebted to Dr. C. A. Thelander, gynæcologist to the Brisbane General Hospital, and Dr. A. D. D. Pye, general superintendent of the Brisbane General Hospital, for their assistance and their permission to report these cases.

### Bibliography.

J. B. DeLee and J. P. Greenhill: "Principles and Practice of Obstetrics", 1943.

J. P. Greenhill: "The 1942 Year Book of Obstetrics and Gynecology. 1942, page 89.

G. F. Melody: "Hydatidiform Mole with Severe Pre-Eclampsia", *American Journal of Obstetrics and Gynecology*, Volume LI, June, 1946, page 901.

C. A. Thelander: Personal communications.

# Reviews.

## PENICILLIN AND ITS PRACTICAL APPLICATION.

Medical literature has been deluged during the past few years with books and papers on penicillin; but a book which has been produced under the general editorship of Sir Alexander Fleming himself represents a complete and authoritative summary of penicillin therapy as it stands today.[1]

The book contains a series of independent contributions by "experienced and eminent men who have worked with penicillin in Great Britain". Their opinions and practical methods differ slightly, and there is some overlapping; but these are not disadvantageous, comparison and contrast lending interest to the reading.

In the first or general section of the book Fleming contributes two chapters, one on the history and development of penicillin, introducing some interesting sidelights in the romance of discovery, the other on the bacteriological control of penicillin therapy. In both chapters the information is set out in meticulous detail and with a clarity and simplicity which can be enjoyed by all readers. Fleming also gives the right perspective to the place of penicillin amongst the antibiotics and lays down the principles of treatment. Both chapters are well illustrated and are the most outstanding in the book. Included in this first section also are chapters on the chemistry and manufacture of penicillin and its pharmacy, pharmacology and methods of administration.

The second section of the book is entirely clinical, giving each author's view on the use of penicillin therapy in a disease or an infection of some particular region of the body. The entire range of penicillin-sensitive conditions is considered in twenty authoritative and clearly written chapters; these contain many references and illustrations. Dental and veterinary diseases are also given fairly full consideration.

The final section is a condensed résumé of much of the preceding chapters and is written for the general practitioner. This chapter is superfluous; it does not contain enough detail to be of much practical value. The book as a whole, however, will undoubtedly be considered a classical contribution to medical literature and is strongly recommended, not only because of the general interest of its topic, but as a reference book on penicillin therapy of hitherto unequalled excellence.

The typography, although conforming to war economy standards, is clear and the paper is good. There is an excellent list of references and the index is satisfactory.

## A HANDBOOK OF PRACTICAL ANATOMY.

The Professor of Anatomy in the University of Oxford, W. E. LeGros Clark, has produced a book on practical anatomy for use by medical students in the dissecting room.[2] Actually it is a revision of a book written over thirty years ago by the late Professors F. G. Parsons and W. Wright, and revised in 1932 under the editorship of the late Professor E. P. Stibbe. However, it is virtually a new book.

The first aim in this volume is to reduce the amount of minute topographical detail that the medical student must learn. Working on the basis that only a small percentage of medical students will ever do major surgery, the author has omitted all factual data whose importance lies solely in their application to specialized operative technique. He has purposely made it a manual for medical students and not for post-graduate students in surgery.

The second aim has been to help the student to correlate his study of dissecting room anatomy with the anatomy of the living body, and to relate structure and function. To this end, instructions for preliminary and supplementary studies at each stage of dissection are provided throughout the book. These should prove of great value to the student.

The text follows in the main the usual dissecting round. After an introductory chapter emphasizing the above points and giving practical instructions in the art of dissecting, the various parts of the body are taken in turn—upper extremity, lower extremity, head and neck, brain, thorax and abdomen. The section on the brain includes a rapid macroscopic staining technique, which will allow the student in a few minutes to stain hand-cut sections with sufficient contrast to enable him to follow most of the important fibre tracts up and down the brain stem and to identify and define most of the important masses of grey matter.

The author has produced a useful manual fulfilling the aims mentioned above. However, we feel that the instructions for dissection are not detailed enough. If the students' dissecting manual is meagre in its instructions, experience shows that the student is continually becoming lost, and fails to learn his anatomy accurately. Accurate knowledge will be the only knowledge of any real use to him later.

The format, type and arrangement of the book are good. The illustrations could be improved. An index is provided.

[1] "Penicillin: Its Practical Application", under the general editorship of Professor Sir Alexander Fleming, M.B., B.S., F.R.C.P., F.R.C.S., F.R.S.; 1946. London, Africa, Australia, Canada, India, New Zealand: Butterworth and Company (Publishers), Limited. 8¼" x 5½", pp. 392, with illustrations. Price: 38s. 9d.

[2] "Practical Anatomy", revised and rewritten by W. E. LeGros Clark, M.A., D.Sc., F.R.S., F.R.C.S.; 1946. London: Edward Arnold and Company. 8¼" x 5½", pp. 486, with many illustrations. Price: 25s.

## GRAY'S ANATOMY.

Although the new twenty-ninth edition of "Gray's Anatomy" has appeared since the cessation of hostilities, its preparation was commenced during the war, and the editors, publishers and all concerned have continued to be harassed by wartime restrictions and commitments.[1] In spite of all difficulties, however, the publication not only maintains its high degree of excellence, but careful revision preserves its reputation as an up-to-date text incorporating recent advances in knowledge.

There are almost 200 new illustrations, including some which replace old familiar originals, and others which have been redrawn in the interest of greater clearness and accuracy. Most of them effect considerable improvements, but we miss the old figures replaced by the new figures 285 and 286, and the excellent arrangement in which certain figures and their keys (for example, figures 309 and 310) appeared on opposite pages. The X-ray plates are brilliant and several of the new figures are particularly good, for example, figure 1001, A and B, of the cervical sympathetic, and figures 1052, 1069 and 1070, which improve the section of the ear. While most sections, however, have new figures which improve their presentation, there is still room for improvement in some of the figures illustrating microscopic anatomy.

While the text, for the most part, is excellent and beyond criticism, it is suggested that certain minor parts could with advantage be rewritten. For example, the student should be able to find a little more help in his understanding of the latest advances and descriptions of the cervical, renal and pelvic fasciæ, both male and female, the fascial spaces of the hand, and special regions of the peritoneal cavity. A few more figures in some cases might effect all that is necessary. Also in the section of myology, it would be useful to find tables classifying, in terms of type of movement produced, the muscles which act on the important joints. Although, too, the topic of the kinds of movement permitted in joints is discussed in a scholarly way on page 444, clearer and more detailed descriptions of the various movements at the important joints might with advantage be included for the benefit of the more junior students.

The section on embryology has received special attention and forms a very valuable, carefully illustrated and succinct account of human development. It is suggested, however, that certain parts of the text still make difficult reading for the average medical student, who is sometimes forced to refer to other texts. There are very few topics in embryology which it is essential for a medical student to know and which cannot be made easy for him to understand. In these days when there is a tendency to reduce the time devoted to anatomy, it is becoming more necessary to assist the harassed student.

With the constant and careful revision to which "Gray's Anatomy" is subjected and which no national emergencies seem to hinder, the book is approaching that degree of perfection which makes it increasingly difficult to fault in any way. In spite of the minor criticisms expressed above, which are based on constant reference to its pages for over a quarter of a century, "Gray's Anatomy" still deserves its position as one of the leading texts familiar to students not only in the English-speaking schools, but in many others throughout the world, and still stands as a monument to the brilliant achievement of three generations of British anatomists.

[1] "Gray's Anatomy, Descriptive and Applied", edited by T. B. Johnston, C.B.E., M.D., and J. Whillis, M.D., M.S.; Twenty-Ninth Edition; 1946. London, New York, Toronto: Longmans, Green and Co. 10" x 6¼", pp. 1618, with 1,359 illustrations, 642 in colour. Price: 70s. net.

The Medical Journal of Australia

SATURDAY, APRIL 5, 1947.

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*References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.*

*Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.*

## THE PHOTOFLUOROGRAPHIC SURVEY AND AFTERWARDS.

GROWING appreciation of the potential value of the photofluorographic survey or "mass miniature radiography" in the control of tuberculosis is reflected in the voluminous literature which now surrounds the method on its technical, administrative and epidemiological sides. New problems are now coming more into focus, especially how to interpret the result of the survey, how to translate it with discrimination into helpful measures of control, and how best to help those who need help without intruding unnecessarily into the lives of many people who do not and causing unnecessary worry and expense to them and to the State. The photofluorographic survey is no longer to be regarded as an end in itself: in fact, it could not now be so conducted without its apparatus inviting comparison with one of those slot machines at travelling sideshows into which Jack and Joan may insert their coin and receive a card telling them their fortunes, good or ill, in plausible language.

In two European countries reports of surveys on a large scale by means of mass miniature radiography have recently been published. These have a much greater value than lay in their original objective of case-finding for military or industrial purposes, because the photofluorographic results have been confronted with the clinical findings: what is more important, it has been found possible to keep in touch for a period of two years with most of the persons who were found to be tuberculous.

The first of these surveys was conducted in the Swedish Navy during the years 1941 and 1942 and has been reported by A. Nelson.[1] The number of persons examined was 44,072, all of whom had been passed as medically fit for naval service: nearly all were between the ages of 15 and 44 years. The number whose photofluorograms showed evidence of disease was 1,210, or 2·7%; but when a check was made of these persons by skiagraphy, in 93 or 8·4% no evidence of pulmonary disease was found, showing, according to Nelson, that in judging photofluorograms there is "a tendency to over-pessimistic diagnosis". Sixty-seven persons whose lungs showed photofluorographic evidence of disease were not further examined "for reasons which may be assumed to be of a purely military nature". When the remainder had been examined it was found that in only 1·05% of the total number of persons examined was the tuberculosis found deemed to be significant: it was active in 0·45% and open in 0·31%. Pneumothorax treatment or thoracoplasty was applied to 104 persons; but improvement was seen in only 60% of these and 8% were already dead. Nelson points out, however, that the mortality was considerably less than the general mortality for persons within the same age groups suffering from "open" tuberculosis. Two hundred and eighty-four persons were observed in hospital for an average of 109 days; the median period of observation was 57 days, but for those suffering from active tuberculosis the average was 254 days and the median 235 days.

The other survey was made in France by A. Bidermann and his collaborators,[1] also in 1941 and 1942 (during the occupation), with the aid of a complex organization which included the use of a special railway carriage. Railway workers were examined to the number of 69,790, of whom 422 were known already to be suffering from tuberculosis. The survey revealed 120 persons suffering from "open" tuberculosis previously unsuspected (0·23% of those examined), 11 with active tuberculosis from whom no bacilli could be recovered (0·02%) and 279 with "pleural or pulmonary abnormalities necessitating phthisiological surveillance", which seems a cautious designation. Collapse therapy was instituted in the case of 70 persons, of whom 11 (or 16%) were already dead at the time of the report. Of those who were kept under surveillance 14% developed manifestations of active tuberculosis during the observation period. In this survey 13,249 of the workers were examined photofluorographically a second time one to two years after the first examination: of these only nine were found to have open or active tuberculosis (though in these for the most part the disease was well advanced) and only 32 (0·24%) were eligible for surveillance. Of the surveillance the authors write:

> *Ce contrôle doit être effectué avec discernement afin d'éviter de créer une psychose de la tuberculose et de provoquer des arrêts de travail injustifiés.*

In this country, which led the way in demonstrating that mass miniature radiography may be applied to the detection of every tuberculous recruit in an entire army, the impetus seems to have been lacking to follow up the photofluorographic survey from the clinical aspect and to coordinate the work in the way demonstrated by our colleagues in Sweden and France. There is no doubt much good work to be done in Australia yet by means of mass miniature radiography. No doubt, too, opportunities will arise out of it for clinical studies as valuable as any that have been reported from abroad, especially when surveys are made of groups of people exposed to extraordinary risk of tuberculosis infection, such for instance as the mental hospital population—though these have no coin to "put in the slot". To offer photo-

[1] *Acta Tuberculosea Scandinavica (Supplementum XVI)*, 1946.

[1] *Revue de la tuberculose*, 1946.

fluorography on a very large scale to the general population in the near future, as has been suggested in some quarters, would seem in the light of the European experience to be an unbalanced project. To balance it we would need to make adequate provision for the accommodation, nursing care and social welfare of those dozens or hundreds who would no doubt be found by X rays and by medical examination to suffer from tuberculosis in a progressive form: in this essential provision we are greatly in arrears and many of our people already known to be tuberculous are dying and slipping beyond hope of recovery while plans to help them remain on paper.

# Current Comment.

## NUTRITION AND ENDOCRINE DISTURBANCES.

Nutrition is one of the subjects certain to be of increasing importance, and the recognition of conditions believed to be due to altered states of nutrition will, so we hope, eventually become simpler. At present the diagnosis of nutritional deficiencies is in many cases far from easy, if our standards approach any reasonable degree of scientific accuracy. Endocrine disturbances are likewise not always clear cut, as the pictures seen clinically are not often those of deficiency or excess of a given secretion, and the interactions of the endocrine systems are complex, introducing an element of doubt or even of guesswork. It would seem, therefore, an ambitious programme to correlate disturbances of nutrition with those of the endocrine organs.

Gerson R. Biskind and Morton S. Biskind have attempted an integration of this kind, and though it is well to be cautious of generalizations, their article gathers together many threads in an interesting fashion with commendable brevity.[1] They point out that for a number of years observations have been accumulating concerning the relationship between alterations in the nutritional state and endocrine disturbances. The isolation of the first crystalline œstrogen led to the finding that this and other related steroids are rapidly inactivated in the body. G. R. Biskind devised a method of studying this problem in the intact animal; in this a pellet of œstrogen was placed in the spleen of a castrate rat. As the steroid was absorbed it had to pass through the liver before reaching the general circulation, and it was found that there was no sign of any œstrogenic effect on the animal, nor of any androgenic effect when an androgen was similarly implanted. The appropriate endocrine effects were, however, noted when the spleen was transplanted after being provided with a collateral circulation. These and many other experiments and observations left no doubt that the liver had the power to destroy œstrogens and androgens. But it was also found that hepatic lesions could be produced by nutritional deficiency, and the same technique was adopted as in the previous experiments to observe the effect of disturbed nutrition on the liver. Castrate female rats were found to display continuous œstrus from the influence of an implanted pellet in the spleen only when the diet was depleted of the vitamin *B* complex. Supply of these elements restored the capacity of the liver to metabolize œstrogen once more. Further work showed that thiamin and riboflavin alone had this power. When these experiments were repeated with androgens nothing happened; in other words no significant impairment of the liver's ability to destroy androgen resulted from a deficiency of thiamin and riboflavin. This led to the concept that excessive uterine bleeding and chronic cystic mastitis were due rather to deficiency in hepatic metabolization of œstrogen than to over-production of this endocrine secretion.

Many other cognate observations are quoted by the authors dealing with changes in the body attributed to excess of œstrogen, or impairment of the œstrogen-androgen balance, in the presence of hepatic insufficiency. For example, an unpublished observation is quoted that hyperplastic proliferation of the uterine lining has been found in aged female subjects dying of cirrhosis of the liver. Cutaneous vascular lesions associated with hepatic cirrhosis have also been found to be related to nutritional deficiency and production of excess of œstrogen. From this evidence it is assumed then that the inactivation of œstrogens in the liver is controlled by various factors which may influence that organ in this particular metabolic function. In the female the effects of retention of excess œstrogen are fairly easy to recognize. In the male it is not so simple, but lessening of testicular function, leading perhaps to infertility and even gynæcomastia, may be seen. The authors state that the latter has also been seen in troops in the Pacific zone who were captured by the enemy and kept for long periods on insufficient diets. Pursuing the matter further, the authors state that they have examined patients suffering from some recognizable clinical state due to excess of œstrogen. In every instance they claim they could demonstrate some form of deficiency of the *B* complex, such as glossitis or cheilosis. These patients were then treated with a diet rich in vitamins, and large doses of the different items of the vitamin *B* complex were given by mouth daily, supplemented in some cases with parenteral administration. It is claimed that over a period of five years good therapeutic results have been obtained. In respect of the symptoms due to excess of œstrogen the response was excellent. Maintenance doses were continued on a lower level, since it is believed that once serious deficiency has occurred the tissues may lose some of their capacity to thrive functionally on the usual requirements.

The authors also deal with some aspects of diabetes from the nutritional standpoint, and claim that striking reductions in the blood sugar levels have been achieved by vitamin therapy. Maybe there is some truth in their contention that vitamin deficiencies are widespread owing to the consumption of more "purified" foods, and worse dietetic habits. At the same time it is hard to credit that such important deficiencies have been common in all communities. Moreover, there may be fallacies in these observations, and one hesitates to draw sweeping deductions. However, it would be strange indeed if important relatfonships did not exist between changes in nutrition and the endocrine system, seeing that the latter has such vital affinities with growth and metabolism. Unfortunately it looks as if quite a considerable part of the world we so proudly call civilized will be a demonstration theatre for deficiencies of diet during the immediate future.

[1] *American Journal of Clinical Pathology*, December, 1946.

## SUDDEN DEATH OF YOUNG PEOPLE.

The occurrence of sudden death in young persons is always distressing. The question always arises whether something might have been done to prevent it, and naturally it is important also to know if the fatal lesion was or could have been discovered during life. An unusual opportunity presented itself during the recent world war to collect details of sudden deaths occurring in an army, apart from the accidents and casualties of war. In the issue of November 23, 1946, attention was drawn in these columns to the sudden deaths of three persons suffering from rheumatic fever, for which no adequate reason could be found. These deaths were reported by G. C. Griffith and R. W. Huntington and occurred among thirteen fatal cases in a series of 7,165 acute rheumatic fever infections in the United States Navy. Alan R. Moritz and Norman Zamcheck have reviewed a large series of sudden deaths, and have published a comprehensive article from the Army Institute of Pathology in Washington and the Department of Legal Medicine of the Harvard Medical School.[1] In this review an examination was made of some 700 to 800 cases as being representative of about 1,000 reports on autopsy

[1] *Archives of Pathology*, November, 1946.

material obtained from American soldiers who had died suddenly. Five principal categories were recognized. These were heart disease, intracranial hæmorrhage, meningococcæmia, miscellaneous diseases of mixed ætiology, and an obscure group in which no satisfactory cause of death was revealed. One curious fact elicited from study of the whole series was that the soldiers who had died suddenly appeared to be heavier in body weight than the average of inductees. Statistical study of the figures, however, casts doubt on the validity of this observation, and it appears that the reason lies either in increase in weight of soldiers as a class after enlistment, or error in the records of weight made at the post-mortem examination, the latter being usually estimates only. It is, of course, necessary to have a clear concept of what is intended by the term sudden death. A pathologist once, when asked whether certain microorganisms would be instantaneously killed by an antiseptic, replied evasively that there were degrees of instantaneous death. Moritz and Zamcheck lay down criteria in their study as follows. The soldiers were under the age of forty; they were ambulatory and presumably not suffering from a dangerous disease at the time of the fatal seizure; the interval of time between the onset of symptoms and death was less than twenty-four hours; and post-mortem examination either revealed a recognizable disease as a cause of death, or else failed to establish such a cause, apart from any considerations of the history. Organic heart disease was responsible for between 200 and 300 deaths. Of these the great majority were due to coronary arteriosclerosis, only some thirty odd being due to other cardiac diseases, such as acute myocarditis, syphilitic aortitis, rheumatic disease, and fibrosis of unknown cause. Both the rheumatic and syphilitic deaths came as a complete surprise, but it is noteworthy that four men included in the former category collapsed unexpectedly during physical exertion. The coronary cases were of particular interest. The incidence of fatal coronary disease rose rapidly with age; only 22% of the cases were drawn from the age groups under thirty. There was no particular relation to body weight. An attempt was made to analyse the figures of those deaths occurring in apparent relation to physical exertion. This is not easy, for it is possible for death to occur during a period of inactivity which might bear some relation to previous exertion. The conclusion was reached that if a young man dies unexpectedly of coronary disease, the onset of the terminal attack of heart failure is more likely to take place during strenuous physical exertion than during sleep. This observation is different from that recorded by Masters, Dack and Jaffe (see THE MEDICAL JOURNAL OF AUSTRALIA, January 11, 1941, page 53). These authors investigated 1,620 attacks of coronary occlusion. They found that 52·2% of the attacks occurred while the patient was asleep or at rest, 21% occurred during routine activity, 15% occurred while the patient was walking at an ordinary pace, and 9% occurred during moderate activity. Only 2% were associated with unusual activity.

In about one-fifth of the cases that are discussed by Griffith and Huntington there had been previous symptoms which could in retrospect be regarded as consistent with cardiac insufficiency, but in the remainder there was no record of anything which might have given rise to suspicion. In practically every instance the main coronary trunks were the seat of severe atherosclerosis, and except for a lesser degree of calcification there was little if any difference between the observed changes and those seen in older people suffering from coronary disease. Intracranial hæmorrhage was the next cause in order of frequency. In more than half of these subarachnoid bleeding occurred from "berry" aneurysms. In a number of autopsies no evidence of these lesions could be found, though the history and findings after death were otherwise indistinguishable. Physical exertion seemed to play no significant part. In the remainder of this group the fatal event was as a rule due to the rupture of intracerebral vessels, though the cause could not be ascertained in some instances. The third important group was that including men who died of fulminating meningococcal infection. Nearly one-third of the men reported as dying of this disease did not live for more than twenty-four hours after the onset. Adequate proof was available of the existence in these men of an acute meningococcæmia. The transition from the prodromal to the fulminant phase of the infection was characteristically abrupt, and the shocked state of the patients gave rise to a diagnosis of the Waterhouse-Frederichsen syndrome in many instances. Over eighty specimens of adrenal glands were reexamined in order to discover their condition, and only in five were the glands thought to appear normal, most of them showing changes of varying degree, while over half were the seat of massive hæmorrhagic necrosis. It is of interest to read, as might be expected, that there were instances in which the existence of meningococcal infection was not suspected at all till an autopsy was performed. The miscellaneous group contained a varied collection. Diphtheria, hepatitis, newgrowths, diabetes, intestinal obstruction, other forms of meningitis, pneumonia, thrombophlebitis, pulmonary tuberculosis and acute pancreatitis figure in this motley list among other causes. Finally there is the mystery group, actually numbering 140 individuals, in whom no satisfactory cause could be discovered. Post-mortem changes were usually agonal in nature, and although some evidences of pathological changes were not very infrequent, such as vascular degenerative affections and enlargement of the heart, the degree of these and the condition of the organs otherwise failed to satisfy the pathologist that he could say what caused death. It will be seen that little can be done in the aggregate to prevent such tragic events, especially when we must admit that we sometimes cannot explain how or why "the blind Fury with the abhorred shears" of whom Milton speaks "slits the thin-spun life".

## THE BIBLIOGRAPHY OF SCIENTIFIC AND INDUSTRIAL REPORTS.

MORE than six months have passed since the first section of the "Bibliography of Scientific and Industrial Reports" was published in this journal. These abstracts are intended to let workers in medical and other branches of science know something of the research that was carried out during the war under the auspices of the Allied Governments. Included with them is information on the results of research that has been gathered from former enemy countries. The abstracts have been prepared by the United States Department of Commerce through its Publication Board, and have been made available throughout Australia by the Council for Scientific and Industrial Research Information Service. No less than twenty-one journals have cooperated with the Council for Scientific and Industrial Research, and up to the present time nearly five thousand abstracts have been published. The officer-in-charge of the Information Service writes that requests for original reports to the number of 931 have been received. Of these reports 128 have been procured from local sources. Sixty-eight of the 128 were obtained by the Secondary Industries Division of the Ministry for Post-War Reconstruction, which deals with reports originating in former enemy countries; and 60 were obtained from the head office of the Council for Scientific and Industrial Research. The remaining 803 reports have been ordered from the United States. So far over 300 of these orders have been filled. The publication of these abstracts has clearly been well worth while.

## INDEX TO "THE MEDICAL JOURNAL OF AUSTRALIA".

THE index to THE MEDICAL JOURNAL OF AUSTRALIA for Volume II, 1946, has been published. Copies will be sent as usual to those on the regular index mailing list. Readers who wish to receive a copy of the index and who have not asked to have their names placed on the list are invited to communicate with the Manager at The Printing House, Seamer Street, Glebe, New South Wales.

# Abstracts from Medical Literature.

## BACTERIOLOGY AND IMMUNOLOGY.

### Modification of Human Red Cells by Virus Action.

F. M. Burnet, J. F. McCrea and J. D. Stone (*The British Journal of Experimental Pathology*, August, 1946) began their study on the modification of human red cells by virus action with an examination of the receptor gradient for virus action in human red cells. Observations made previously had established that viruses of the influenza group could be arranged in a series so that cells treated by a given virus failed to be agglutinated by strains earlier in the series, but were still susceptible to those later in the series. A new approach to this fact came from the discovery that after treatment with virus, red cells became agglutinable by human and other sera previously without action on them. Human red cells of group O which had been washed and centrifuged were treated with allantoic fluid virus for thirty minutes and then washed and resuspended in saline solution. They were then tested for agglutination by a single dilution of a series of virus samples including that used on the cells originally, and a symmetrical result was obtained, mumps and Newcastle disease virus being at the left end of the scale, and swine influenza and two strains of influenza B at the right. Here then was a reason for cells treated by swine influenza being susceptible to a large number of other viruses, and agglutinable by a large number of sera, in dilutions not related to the antibody present. Further experiments showed that receptors could be removed from red cells by enzymes such as the lecithinase of *Clostridium welchii* toxin A or by filtrates from cholera cultures. It appeared that other viruses were more effective in removing mumps virus receptors than was enzyme action, while filtrates from vibrios could remove very largely the cell receptors, leaving very little agglutinability in the cells by other sera. Problems arising from this work are twofold: (*a*) it is possible that enzymes play an essential part in the invasion of susceptible cells by virus, as well as in the adsorption onto red cells; and (*b*) the relationship to chemotherapeutic studies may be a profitable line of approach to the problem of virus disease.

The second paper in the series is by F. M. Burnet and S. G. Anderson (*ibidem*) on the agglutination of modified human red cells by sera from cases of infectious mononucleosis. Newcastle disease virus stands early in the series of viruses which render human cells susceptible to those later in the list, and cells so treated will agglutinate to some extent with a large proportion of normal human sera. On account of the existence of a small epidemic of infectious mononucleosis, samples from patients whose sera showed a heterophile antibody titre became available for testing, and it was found that these agglutinated cells treated with Newcastle disease virus. An experiment was designed to see if any difference existed between virus hæmagglutinin and the cell sensitizing agent of the virus, and this was found to be clear cut, with repeated doses of cells, the sensitizing agent became less, while the hæmagglutinating power diminished only a little. The authors suggest that these observations may lead to the identification of the agent responsible for the syndrome of infectious mononucleosis, or that the phenomenon observed may be due to incidental immunological and chemical similarity between substances concerned in the reaction.

### "Q" Fever.

A whole issue of the *American Journal of Hygiene*, that of July, 1946, has been devoted to a series of papers on "Q" fever in the Mediterranean area, and in troops who served in that area. The papers are written by members and ex-members of the United States Army, authorized by the Commission on Acute Respiratory Diseases. The investigation centred about outbreaks of "atypical pneumonia" beginning in Athens, from which a rickettsia was isolated; later another developed in northern Italy, and finally in disembarkation ports and camps in Virginia where troops from Italy were in transit. A case was found in Panama, and infections occurred amongst members of the laboratory personnel concerned. The clinical features of the disease, described by F. C. Robbins and C. A. Ragan, include high fever, acute headache, together with X-ray evidence of pulmonary consolidation, and a considerable loss of weight. Laboratory tests revealed complement fixing antibodies and agglutinins in the blood of convalescents, and rickettsiæ were recovered from guinea-pigs injected intraperitoneally with the blood of patients, on sixteen occasions out of twenty attempts. The epidemiology is discussed by F. C. Robbins, R. L. Gould and F. B. Warner. Some evidence of endemicity was obtained in some Italian villages and some explosive outbreaks were described as "place" infections, occurring only in certain billets, and the finding of mites as a possible vector is considered, although no actual proof is forthcoming. The incubation period appeared to be fourteen to twenty-six days. The ætiological agent is discussed by F. C. Robbins, R. Rustigian, M. J. Snyder and J. E. Smadel. Primary isolation was by the intraperitoneal inoculation of 3·0 to 5·0 millilitres of whole blood into guinea-pigs, which developed fever about the ninth day, and also by the inoculation of serum into developing eggs. One isolation was made from cerebro-spinal fluid. Guinea-pigs did not die from primary infection, but developed acute disease after inoculation with yolk-sac cultures. The yolk-sac culture of one particular strain was also found to give emulsions which were effective antigens for complement fixation tests. Serological comparison between strains isolated from Australian patients and these Italian strains indicated that they were identical. F. C. Robbins and R. Rustigian describe a laboratory outbreak of twenty cases, and emphasize that the handling of infected eggs appeared to be more dangerous than the handling of experimental guinea-pigs. Six further papers describe clinical and epidemiological features of the Camp Patrick Henry outbreak and of sixteen cases in the laboratory at Fort Bragg, some of which were extremely severe and in four of which relapse occurred. Again emphasis is laid on the infectiveness of yolk-sac cultures. The final paper by Norman H. Topping, Charles A. Shephard and Robert J. Huebner describes an immunological comparison of strains and compares Australian, American, Balkan, Italian and Panama strains. Complete reciprocal cross immunity was established in guinea-pigs, but considerable differences in sensitivity of antigens prepared by ether extraction of yolk-sac cultures appeared. The authors were satisfied that the strains were identical with *Rickettsia burneti*, but could not establish a vector for the disease.

### Biological Investigations on Proactinomycin.

H. W. Florey, M. A. Jennings and A. G. Sanders (*The British Journal of Experimental Pathology*, October, 1945) have made a detailed investigation of the biological properties of proactinomycin, a product of *Proactinomyces gardneri*, first described by Gardner and Chain in 1942. Pharmacological tests showed that it slowed the heart rate and increased the blood pressure of cats, that a 1 in 1,000 dilution killed leucocytes in half an hour, while a 1 in 16,000 solution had no effect at all on white cells. Fourteen milligrammes placed in the stomach of mice killed them in one and a half hours. Small doses given over a period of five days did not cause symptoms in mice, and histologically only a small amount of cloudy swelling in the liver was seen. It was absorbed from the stomach, and could be detected in the liver, but not in the blood serum, and a fair amount was present in the urine. Antibacterial tests showed activity in high dilution against cocci and Gram-positive bacilli, weak activity against Gram-negative bacilli and tubercle bacilli. The presence of whole blood reduced its activity when tested by the plate and cylinder method. No resistance by staphylococci to the substance could be detected during the experiments and penicillin-resistant staphylococci were not resistant to proactinomycin. Tests in mice given hæmolytic streptococci intraperitoneally showed considerable protection exerted when the drug was given by mouth, and doses much smaller than anticipated proved effective. The authors suggest that the drug may be of use in the laboratory or in treatment of severe infections the causative organisms of which had become penicillin-fast.

### Studies on Malarial Parasites.

J. W. Ferrebee, J. G. Gibson and W. C. Peacock (*The Journal of Infectious Diseases*, May-June, 1946) have recorded some observations regarding the age of the erythrocyte invaded by *Plasmodium vivax*, following an earlier work suggesting that the different composition of the young red cell might influence the choice by the parasite of its host cell. The principle of the method employed was that radioactive iron administered intravenously appears within twenty-four hours in the hæmoglobin of newly formed red cells, and the radioactivity of the blood can therefore be used as an index of the rate of formation of cells and their addition to the blood stream. A patient under treatment with malaria was also given a dose of radioactive

iron, parasite counts and radioactivity of the blood were estimated daily, and the radioactivity of parasitized red cells had a radioactivity fifteen to fifty times greater than that of waste blood. The authors discuss possible arguments against this suggestion that *Plasmodium vivax* selectively invades young erythrocytes.

### Organisms Resembling Pleuro-Pneumonia Organisms and Arthritis.

R. WALLERSTEIN, B. VALLEE AND L. TURNER (*The Journal of Infectious Diseases*, September-October, 1946) have investigated the possible relationship of the organisms resembling those of pleuro-pneumonia to Reiter's disease, rheumatoid arthritis and ulcerative colitis, by testing sera from patients against an antigen of the living particles obtained from washing culture plates. Titres of below 1/16 were ignored. Samples from 102 patients were tested. Thirty-two normal control sera were below the critical dilution, as also were 36 samples from patients with uncomplicated ulcerative colitis and 20 from cases of rheumatoid arthritis. Four sera showed significant titres, two from patients who had the triad of Reiter's syndrome, conjunctivitis, arthritis and urethritis. A detailed review of literature on the subject of organisms resembling those of pleuro-pneumonia is included.

## HYGIENE.

### The Use of Chlorine Dioxide in Water Treatment.

G. P. VINCENT, J. D. MACMAHON AND JOHN F. SYNAN (*American Journal of Public Health and the Nation's Health*, September, 1946) state that chlorine added to waters containing phenolic wastes intensifies the unpleasant odours and tastes by producing "chlorphenols". Chlorine dioxide destroys both these and their taste. Chlorine dioxide is a yellow to red gas, liquid at 10° C., best produced by the action of chlorine on sodium chlorite. Chlorine dioxide has more than twice the oxidizing power of chlorine, and though in itself unstable, it can readily be obtained from sodium chlorite *plus* chlorine. Pre-chlorination of the water *plus* chlorine dioxide is now in use.

### Pulmonary Tuberculosis.

P. ARNECULLE AND G. CANETTI (*La presse médicale*, June 29, 1946) describe the multiplication and destruction of tubercle bacilli in tuberculosis of the lungs. Five hundred lesions were studied. It was found that the tubercle bacilli were most numerous in the caseous borders of tuberculous cavities. This explained the prolonged duration of cavities and the risks of spread of infection in such cases. With cavitation the bacilli are often numerous and persistently present in the sputum. Hence the importance of the treatment and cure of patients with cavities in order to prevent the spread of the disease.

### Pertussis Immunization in Edmonton.

G. M. LITTLE (*Canadian Journal of Public Health*, August, 1946) reports that by the use of pertussis vaccine, later combined with diphtheria toxoid, a reduction in the three-yearly pertussis peak was obtained. In the three years 1943-1945, 4,744 children were immunized; 56 contracted the disease and only 11 of them had received three doses of the vaccine, 2·7 per thousand. Of 14,300 children not immunized, 964 were attacked, an incidence of 67 per 1,000. The author favours the combined diphtheria-pertussis-tetanus vaccine in four one-millilitre doses.

### The Modern Treatment of Syphilis.

EVAN W. THOMAS (*American Journal of Public Health and the Nation's Health*, September, 1946) states that in early syphilis aqueous penicillin, 20,000 units, is given every three hours for at least sixty doses over seven and a half days. In neurosyphilis the time must be lengthened to nineteen days, 40,000 units being given every three hours for 150 doses. At Bellevue in the seven and a half days' treatment penicillin is combined with eight daily injections of 0·04 gramme of "Mapharsen". Serological tests are an essential check. Ambulatory treatment with penicillin in beeswax and peanut oil is being tested, 600,000 units being given a day for eight days.

### Recent Developments in Murine Typhus Fever Control.

JOHN S. WILEY (*American Journal of Public Health and the Nation's Health*, September, 1946) states that over 5,000 cases of rat-borne typhus were reported, and that 67% of them occurred in nine southern and south-eastern States. The disease seldom occurs north of 33° north latitude. Rat proofing and rat eradication from business premises followed up by strict inspection can reduce the incidence of typhus by 50%. Garbage, storage and disposal, storage of chicken feed, correct stacking of goods and cutting down harbourage are of value. Two new rat poisons have been helpful. The best was a solution of sodium fluoroacetate, 12 to 14 grammes per gallon. It, however, can kill dogs and cats. "ANTU"—alpha-naphthyl-thiourea—does not readily kill larger animals. Keeping down the intermediate vector is invaluable. Dusting with 10% "DDT" in talc is favoured for runs and other frequented places. Cats and dogs are also dusted and dusting is carried out around foodstuffs by means of a small or large handshaker or a hand pump. Calcium cyanide in burrows kills both rats and fleas. "DDT" produced a reduction of 85% among rat fleas on rats for three months after dusting. Heart blood tests were also made for complement fixation and showed that the percentage of infected rats was greatly reduced in the treated area.

### Trichinosis.

H. B. SHOOKOFF, W. B. BIRNKRANT AND M. GREENBERG (*American Journal of Public Health and the Nation's Health*, December, 1946) describe an outbreak of trichinosis in New York city, and pay particular attention to the value of intradermal and precipitin tests. During February and March, 1945, New York city experienced the largest outbreak of trichinosis in the history of its health department. Eighty-four persons became ill with the disease from eating pork products supplied by a single wholesaler. Among the eighty-four cases there were no fatalities. The commonest type of onset was with œdema of the eyelids and fever. Gastro-intestinal symptoms occurred in a minority of the cases and the onset of such symptoms was usually simultaneous with or subsequent to the onset of œdema. The classical prodromal gastro-enteritis occurred only in seven cases. It is usually stated that the intradermal test gives a positive result in trichinosis during the third week after infection. It is further stated that the result of the precipitin test becomes positive at about the end of the fourth week of infection. The authors state that these views give a false impression of the relative value of the test. In the outbreak reviewed by them the precipitin test gave a positive result earlier in the illness than did the intradermal test. They found that in the second week the precipitin test was far the more likely of the two to give a positive result. In the later weeks of illness the result of precipitin test was in general more often positive than the result of the skin test.

### A Study in "B.C.G." Vaccination.

R. C. FERGUSON (*Canadian Journal of Public Health*, November, 1946) reports a study of vaccination with *Bacille Calmette Guérin* vaccine carried out by the National Research Council of Canada. It was thought that the efficacy of "B.C.G." vaccine would be tested if it was used for the vaccination of unavoidably exposed persons in the environments of hospitals and sanatoria who had not reacted to tuberculin at the time they entered the environment. In the hospital group 2,042 nurses were accepted for study. In the hospital environment the rate of infection was approximately 12% *per annum* among the non-vaccinated who had not reacted to the tuberculin test and the duration of exposure was 2·42 years. The number of cases of manifest tuberculosis that developed in this group following "B.C.G." vaccination was a fourth of what it had been without vaccination. The ratio of vaccinated non-reactors to tuberculin compared with the non-vaccinated non-reactors to tuberculin was 1 to 4·27. In the sanatorium group 1,206 persons were accepted for study. In the sanatorium environment the rate of infection was approximately 60% during the first year of exposure among the non-vaccinated who had not reacted to tuberculin and the duration of exposure was 1·44 years. The number of cases of manifest tuberculosis that developed among this group was a fifth of what it had been without vaccination. The ratio of vaccinated non-reactors to tuberculin compared with the non-vaccinated non-reactors to tuberculin was 1 to 5·03. These findings had statistical significance. The author concludes that "B.C.G." is not a 100% effective prophylactic—its protection is very considerable, but by no means absolute. "B.C.G." vaccination is safe. When the vaccinated and the non-vaccinated non-reactor to tuberculin were compared in regard to the severity of manifest tuberculosis that developed among them it was found that the lesions in the vaccinated group were less extensive than the lesions in the non-vaccinated group. Since the introduction of "B.C.G." vaccination the nursing schools in Saskatchewan (where this investigation was made) no longer have anxiety and worry about the excessive development of tuberculosis among the members of their staffs who do not react to tuberculin.

# Bibliography of Scientific and Industrial Reports.[1]

## THE RESULTS OF WAR-TIME RESEARCH.

*During the war a great deal of research was carried out under the auspices of the Allied Governments. It has been decided to release for general use a large proportion of the results of this research, together with information taken from former enemy countries as a form of reparations. With this end in view, the United States Department of Commerce, through its Publication Board, is making a weekly issue of abstracts of reports in the form of a "Bibliography of Scientific and Industrial Reports". This bibliography is now being received in Australia, and relevant extracts are reproduced hereunder.*

*Copies of the original reports may be obtained in two ways: (a) Microfilm or photostat copies may be purchased from the United States through the Council for Scientific and Industrial Research Information Service. Those desiring to avail themselves of this service should send the Australian equivalent of the net quoted United States price to the Council for Scientific and Industrial Research Information Service, 425, St. Kilda Road, Melbourne, S.C.2, and quote the PB number, author's name, and the subject of the abstract. All other charges will be borne by the Council for Scientific and Industrial Research. (b) The reports referenced with an E number may be obtained in approved cases without cost on application to the Secondary Industries Division of the Ministry of Post-War Reconstruction, Wentworth House, 203, Collins Street, Melbourne, C.1. Copies of these are available for reference in public libraries.*

*Further information on subjects covered in the reports and kindred subjects may be obtained by approaching the Council for Scientific and Industrial Research Information Service, the Secondary Industries Division of the Ministry of Post-War Reconstruction, or the Munitions Supply Laboratories (Technical Information Section), Maribyrnong, Victoria.*

PB 22972. BLACK, A. P., *et alii.* Patch testing (irritation and sensitization). (Bureau of Medicine and Surgery Res. Project X-180, Reports 1 to 4.) July, 1943. 20 pp. Price: Microfilm, 50c.; Photostat, $2.00.

This report gives a summary of the results of tests carried out on human and animal subjects with regard to irritation and sensitization caused by flame-proofing compounds, "Devex Resins", camouflage ponchas, anti-oxidant oils, and corrosion preventive oils. This report is from the National Naval Medical Centre, Bethesda, Maryland.

PB 22955. HOUSTON, C. S., *et alii.* Evaluation of emergency breathing procedure at 25,000 feet. (Bureau of Medicine and Surgery Res. Project X-343, Report 1.) July, 1944. 31 pp. Price: Microfilm, 50c.; Photostat, $3.00.

This is a report of the Altitude Training Unit, U.S. Naval Air Station, Miami, Florida. Instructions were given in emergency breaking procedure (EBP) at sea level, followed by practice and test runs in the low pressure chamber at simulated altitudes of 25,000 feet. A total of 120 man runs are reported. It was found that EBP can increase the oxygen saturation of the blood and thereby prolong consciousness in subjects breathing air at 25,000 feet. EBP is difficult to learn and difficult to perform except in the most favourable circumstances. The ability of many subjects to perform EBP after they have become anoxic from a period of normal breathing is impaired to a point where no benefit is obtained from the method. Subjects have difficulty in performing EBP while attempting even simple tasks and tend to sacrifice EBP to complete their task; severe anoxia ensues. Consideration of the advantages and disadvantages of EBP indicates that it is not a practical procedure for the use of aviation personnel. In part 2 of the study the effects of EBP and hyperventilation were compared. The benefits obtained from EBP while breathing air at 25,000 feet are no greater than those obtained from hyperventilation alone. Hyperventilation performed at the rate of twelve to fifteen breaths per minute is easier and less fatiguing than EBP at the same rate. Five tables, sixteen graphs and three appendices are attached.

PB 22956. HOUSTON, C. S., *et alii.* Evaluation of hyperventilation of ambient air at 25,000 feet. (Bureau of Medicine and Surgery Res. Project X-343, Report 2.) August, 1944. 9 pp. Price: Microfilm, 50c.; Photostat, $1.00.

This is a report of the Altitude Training Unit, U.S. Naval Air Station, Miami, Florida. Object of the study was to determine the value of hyperventilation as a survival technique while breathing air at 25,000 feet. On nineteen of the forty runs (48%) subjects were in good condition at the time the masks were replaced. On twenty-one of the forty runs (52%) acapnia or anoxia required replacement of the mask prior to the required time. Oximeter readings indicate that most, if not all, of the increased arterial oxygen saturation obtained in emergency breathing procedure is due to the increased pulmonary ventilation rather than to the exertion of pressure. The exertion of pressure of some degree apparently is effective in decreasing the incidence of acapnia. If acapnia can be prevented by some modification of hyperventilation this procedure will be of more practical value, since it is easier to learn and easier to teach. A bibliography, a table and two graphs are attached.

PB 25494. KABAT, ELVIN A., *et alii.* A study of the purification and properties of ricin. No date. 20 pp. Price: Microfilm, $1.00; Photostat, $2.00.

Highly toxic hæmagglutinating preparations of ricin can be obtained by fractional precipitation with sodium sulphate. These products are electrophoretically, ultracentrifugally and immunochemically homogeneous and identical in these respects with crystalline ricin, but are only two-thirds as toxic. The toxic and non-toxic forms can be separated by crystallization. Immuno-chemical and toxicity assays of crude extracts of castor beans indicate that these contain both forms of ricin. Ricin was found to have an isoelectric point of about 5·2 to 5·5 and a molecular weight by sedimentation and diffusion of about 77,000 to 85,000 for an assumed partial specific volume of 0·75. References and tables are attached. This work was done under NDRC contract at Columbia University.

PB 22977. LEE, R. H. Determination of effect on dark adaptation of varying intensities of illumination in ready rooms. (Bureau of Medicine and Surgery Res. Project X-162, Report 1.) June, 1943. 11 pp. Price: Microfilm, 50c.; Photostat, $1.00.

Previous tests have shown that red, dark adapter goggles are adequate to preserve dark adaptation in lighted rooms. This report is concerned with the tests which were made to determine the effectiveness of the goggles for the restoration of the dark adaptation once lost. Tests indicated that an illumination of ready room at an average of 20 to 30 foot candles combined with the wearing of goggles for a minimum of 45 minutes to insure a state of proper dark adaptation would make it possible for an aviator to have his night vision for fighting within two minutes after leaving the ready room. Tables and curves record test data. For later findings see PB 22978. This study was made at the National Naval Medical Centre, Bethesda, Maryland.

PB 22978. LEE, R. H., *et alii.* Determination of effect on dark adaptation of varying intensities of illumination in ready rooms: newly discovered fluctutions of periodic nature occurring in dark adaptation thresholds. (Bureau of Medicine and Surgery Res. Project X-162, Report 2.) September, 1943. 5 pp. Price: Microfilm, 50c.; Photostat, $1.00.

This report records the discovery of a phenomenon which makes necessary a reconsideration of the conclusions which were stated in report number 1 of this project (PB 22977). This phenomenon consists of a wave-like fluctuation with a three to seven minute period and an irregular amplitude of from 0·05 to 0·25 log unit test brightness. It has been found in all dark adaptation thresholds below the highest levels. When these fluctuations are taken into account and comparisons of data are made by superposition of the individual curves, it becomes apparent that no advantage is to be gained by wearing dark adapter goggles longer than the thirty minutes now recommended. Ready rooms should continue to use illumination of 20 to 30 foot candles. If all red light is used the intensities should be between 2·5 and 3·8 foot candles. This study was made at the National Naval medical Centre, Bethesda, Maryland.

PB 22984. LEE, R. H. Exploratory project on growth curve of light adaptation. (Bureau of Medicine and Surgery Res. Project X-142.) April, 1943. 4 pp. Price: Microfilm, 50c.; Photostat, $1.00.

The state of adaptation of the eye to the dark depends on the time of exposure to a light source as well as the brightness of the source. On this basis recommendations are made for the procedure to be followed in further studies on adaptation of the eye to the dark. The figures and tables mentioned are lacking in this copy. This document is from the National Institute of Health, Bethesda, Maryland.

PB 22959. LOZNER, E. L. Studies on liquid plasma during second year of storage. (Bureau of Medicine and Surgery Res. Project X-105, Report 1.) July, 1943. 6 pp. Price: Microfilm, 50c.; Photostat, $1.00.

This is a report of the National Naval Medical Centre, Bethesda, Maryland. Preliminary chemical findings on

[1] Supplied by the Information Service of the Council for Scientific and Industrial Research.

eighteen samples of plasma preserved in the liquid state for 15 to 24 months at room temperature are presented. During storage of such duration, the non-protein nitrogen content, the amino acid nitrogen and the "residual" non-protein nitrogen all appear to increase slightly. No chemical changes can be detected in the total protein albumin or globulin content, but it appears from the increase in non-protein nitrogen and amino acid nitrogen content that from 0·5% to 1·0% of the protein is hydrolysed. Therefore, 99% to 99·5% of the precipitable protein can be said to remain intact from the chemical point of view. Two electrophoretic patterns on plasma 15 and 24 months old were within normal limits. When plasma is prepared by a "closed" system with scrupulously aseptic technique, its expiration date when preserved in the liquid state at room temperature in a moderate climate may be extended from the present limit of one year to eighteen months and probably to two years. One table is attached.

PB 22662. MASSACHUSETTS INSTITUTE OF TECHNOLOGY. DEPARTMENT OF BIOLOGY. The X-ray diffraction patterns of normal, deaminated, and partially deguanylated kangaroo tendon. No date. 2 pp. Price: Microfilm, 50c. Photostat, $1.00.

The X-ray diffraction patterns examined both without electron staining and with Calgon or phosphotungstic acid stains show definite alterations in relative order intensities. The most significant changes are in the neighbourhood of the fifth to seventh orders.

PB 23068. PFEIFFER, C. C., *et alii*. Studies on the possible toxicity of sea water desalinated with Permutit "BaH" briquets. (Bur. of Medicine and Surgery Res. Project X-127, Rept. 12.) August, 1945. 5 pp. Price: Microfilm, 50c.; Photostat, $1.00.

Both the precipitated salts resulting from the use of Permutit "BaH" briquets and the desalinated sea water have been assayed for possible toxic effects in growing rats. It was concluded that small amounts of the residue resulting from the Permutit "BaH" desalination of sea water can be ingested without producing evidence of intoxication. Desalinated sea water as a sole source of fluid is non-toxic except for a moderate laxative effect. Test data are included in the report.

PB 22961. RILEY, R. L., *et alii*. Hyperventilation breathing air as a means of maintaining consciousness at 25,000 feet. (Bur. of Medicine and Surgery Res. Project X-454, Rept. 1.) September, 1944. 6 pp. Price: Microfilm, 50c.; Photostat, $1.00.

This is a report of the Naval School of Aviation Medicine, U.S. Naval Air Training Bases, Pensacola, Florida. Fifty male subjects were instructed briefly in the procedure of hyperventilation and then were exposed to a pressure altitude of 25,000 feet in the low-pressure chambers while breathing air. Twenty-four (48%) remained "conscious" for fifteen minutes; twenty-six (52%) required added oxygen before fifteen minutes had elapsed. The minute respiratory volume approximated twenty to thirty litres. Many symptoms of acapnia were noted; none were serious. A bibliography and one graph are attached.

PB 23439. ROBERTSON, A. F., AND KORTY, V. M. Safety of divers during countermine test work. Report of test of October 6, 1942. (Bur. of Medicine and Surgery Project X-70; Naval Ordnance Lab. Memo. 2481.) October, 1942. 5 pp. Price: Microfilm, 50c.; Photostat, $1.00.

Ten and fifty-three pound charges of nitramon were detonated at various distances from submerged divers. The closest ten pound charge (at 460 feet) was found to be audible, but without pressure effect. This corresponds to an instantaneous peak pressure of about fifty pounds per square inch. A fifty-three pound charge at 1,100 feet was merely audible, but at 980 feet the pressure effect was quite noticeable. This pressure effect also corresponds to an instantaneous peak pressure of about fifty pounds per square inch. For continuation of these experiments see PB 23440 by Corey, E. L.

PB 25229. SUGAR, OSCAR. Collapse reactions due to lowered barometric pressure. (A.A.F. A.M.C. Eng. Div. Memo. Rept. TSEAA-696-101.) April, 1946. 10 pp. Price: Microfilm, 50c.; Photostat, $1.00.

This report is a summary of available data and opinion on collapse reactions due to lowered barometric pressure or anoxia, the prevention of such collapse, and treatment when collapse occurs.

PB 23037. TURNER, J. MCCULLOUGH, *et alii*. Toxic gases from assisted take-off units (JATO). (Bur. of Medicine and Surgery Res. Project X-463, Rept. 1.) October, 1944. 6 pp. Price: Microfilm, 50c.; Photostat, $1.00.

The report describes experiments to evaluate the possible toxic hazards to personnel exposed to gases and smoke from assisted take-off units (JATO). Gross examination of guinea-pigs, exposed to the exhaust 22 to 50 feet from the unit nozzle, and microscopic examination of their lungs revealed no acute changes from the normal. The blood of one guinea-pig was found to contain a negligible amount of carbon monoxide hæmoglobin. The short time of exposure (less than one minute) prevented injury from the relatively high CO and HCl concentrations. It was concluded that a more accurate evaluation could be made only by actual tests aboard an aircraft carrier. This work was done by the Naval Medical Research Institute, National Naval Medical Centre, Bethesda, Maryland.

PB 23402. U.S. ENGINEER BOARD. Efficiency of standard army water purification equipment and of diatomite filters in removing cysts of *Entamœba histolytica* from water. (Rept. 834.) July, 1944. 77 pp. Price: Microfilm, $1.00; Photostat, $6.00.

The tests covered by this report consisted of introducing a predetermined number of cysts of the NRS strain into the raw water, passing this water through filters, and collecting samples of the effluent at regular intervals for microscopic examination. Chemical and bacterial analyses of the influent and effluent of a number of tests were made as an additional control. The equipment used consisted of U.S. Army Portable Water Purification Units, Model 1940, and diatomite filters. It was found that diatomaceous silica filtration is much more effective than sand filtration for the removal of amœbic cysts. Design features, being essential for best results, are indicated. The combination of pretreatment, sedimentation and filtration gives results considerably better than filtration alone. Eight photographs, forty-five test results data sheets and two graphs are attached.

PB 22966. WEBSTER, STEWART H., *et alii*. An interim report on the stibine study. (Bur. of Medicine and Surgery Res. Project X-166.) June, 1944. 41 pp. Price: Microfilm, 50c.; Photostat, $3.00.

This is a report of the National Institute of Health, Bethesda, Maryland. The object of this part of the stibine investigation was to determine the toxic effects of antimony hydride or stibine, using several species of laboratory animals as experimental subjects. This highly toxic gas acts as a secondary irritant, affecting chiefly the deeper structures of the lungs and producing congestion, œdema, or even hæmorrhage. Death may result fairly quickly. With some animals the irritant properties can be noted from the watering of the eyes, rubbing of the nose and/or mouth, running of the nose, salivation or even râles. The secondary action appears to be due chiefly to the action of antimony hydride and/or its decomposition products on the blood. The effect of this hæmolytic agent is considered with reference to (1) change in shape and number of the erythrocytes, (2) hæmoconcentration, (3) alteration in the leucocytes, (4) change in hæmoglobin and hæmatocrit values, (5) occurrence of hæmolysis, (6) cell fragility, (7) action of antimony hydride on red cells both *in vivo* and *in vitro*, and (8) miscellaneous measurements, such as coagulation time, icteric index and prothrombin time. The experimental findings on the urine specimens are described with reference to the occurrence of hæmoglobin, albumin, urobilin, urobilinogen and methæmoglobin. The effect of the agent on urine excretion and the experimental work with acid diets are mentioned. The relationship of bilirubin formation to that of hæmolysis in the experimental animals is discussed. The chief pathological findings in the lungs, liver, kidneys and spleens of the animals examined are described. Comparison of acute and chronic toxicity on a few animals suggests that some tolerance to antimony hydride may be acquired rather rapidly. It is also apparent that the agent may stimulate erythropoiesis.

PB 22292 GERMANY. REICHSFÜHRER SS. AMT "AHNENERBE". *Wehrwissenschaftliche Zweckforschung*. (Correspondence of Dachau and Natzweiler concentration camps on procurement of material for research work.) (ALSOS Mission DCC 1.) 1942-1945. 168 pp. Price: Microfilm, $2.00; Photostat, $12.00.

This file contains correspondence in German on the procurement of equipment and supplies for research purposes at the Dachau and Natzweiler concentration camps. Included are decrees from Heinrich Himmler authorizing the use of the facilities of the concentration camp for supporting anatomical studies by Dr. A. Hirt, of the Reich University of Strasbourg. Hirt was given an assignment by the Reichsforschungsrat to find out how living organisms will be changed under the influence of chemical warfare agents, using inmates as experimental material. Secret experiments utilizing human bones are also mentioned.

PB 20503. HAAGEN, E. *Forschungsaufträge: Influenza-gelbfieber-fleckfieber*. (Correspondence on German Air Force medical research projects.) (ALSOS Mission, Haagen 2.) 1942-1944. 381 pp. Price: Microfilm, $4.00; Photostat, $26.00.

This report consists of correspondence and miscellaneous papers concerning research projects carried on for the German Air Force by Dr. E. Haagen, director of the Hygienisches Institut of Strasbourg University and staff surgeon in the German Air Force, and having as their object the production of vaccines for influenza, typhus and yellow fever. In German.

# Medical Societies.

## MELBOURNE PÆDIATRIC SOCIETY.

A meeting of the Melbourne Pædiatric Society was held on October 9, 1946, at the Alfred Hospital, Melbourne, Dr. A. P. Derham, the President, in the chair.

### Patent Ductus Arteriosus.

Dr. H. Lawrence Stokes showed a female patient, aged five years, and a male patient, aged eleven years, both of whom had been successfully operated on for patent *ductus arteriosus*. He also showed a third patient with the same lesion, a female, aged eleven years, for whom operation was contemplated. Those present were invited to listen to the magnified cardiac sounds through multiple stethoscopes attached to a common transmitter, an apparatus specially arranged by Dr. Morris C. Davis.

Dr. C. J. Officer Brown said that the whole problem had many interesting facets. Little was known of the life history of the patients; a number of cases were complicated by bacterial endocarditis or congestive cardiac failure. No considerable series had been followed to a conclusion, and the only evidence was derived from autopsy findings. Dr. Brown said that Keys and Shapiro in 1942 reviewed the literature, obtaining sixty post-mortem reports to which they added a small number of their own. They found that of the cases reported, in 80% death had occurred as a result of *ductus arteriosus*. Endocarditis accounted for 40% and congestive cardiac failure for 30%. A few patients fell victim to aneurysm of the ductus and other lesions. Keys and Shapiro also observed that any patient with *ductus arteriosus* who lived to the age of seventeen years had an average expectation of life of thirty-five years. It was true that some lived longer than this, the oldest being aged fifty-eight years. Thus it could be said that, to judge by the figures, the expectation of life in *ductus arteriosus* was considerably less than normal.

Dr. Brown went on to say that two classes of subjects had been separated—those in whom the condition was well compensated and those with impaired heart function. Those in whom the condition was compensated would have a high diastolic blood pressure, and as the leak increased the diastolic pressure would become lower and lower. The problem was when to operate and how patients should be selected. It was said that if endocarditis had developed they should be submitted to operation. This was before the introduction of penicillin; endocarditis could be controlled with this drug. Dr. Brown said that he had personally ligated the duct in two cases, in both of which endocarditis was present, and in each case the blood was made sterile by penicillin therapy before operation. Even with penicillin, bacterial endocarditis remained an indication for operation. Penicillin should be used before operation. Dr. Brown said that some patients progressed to cardiac decompensation, cardiac enlargement and symptoms of cardiac failure. The only chance in these cases was operation. One of his patients had been approaching this stage; after operation he was dramatically restored to normal cardiac function. Dr. Brown also referred to the child who was underdeveloped. When the leak was small, very little change occurred in the patient's physique. Other patients were diminutive. These children developed in a striking fashion after operation. These three groups provided definite indications for operation. Some authorities stated that only such patients should be treated. Gross, indeed, at first adopted this attitude, but later changed his plans and operated on all children afflicted with patent *ductus arteriosus*. Jones in Los Angeles had operated on sixty patients without a death, so the operation was not a hazardous procedure. Jones ligated every patent *ductus arteriosus* in children, because the risk was so slight and the operation was so trivial. In the case of adults he acted differently. If the patient was aged over twenty-five years, and suffered no disability from the ductus, he refrained from operation unless decompensation or endocarditis supervened. Dr. Brown said that he fell in with this view. In the first 150 cases reported, 107 patients had an uncomplicated patent *ductus arteriosus* and 33 had endocarditis. Cure had apparently been effected in 81 cases. Ten patients had died, six of these deaths being due to hæmorrhage. There was a risk of a tear in the ductus or in the right pulmonary vein. Of the 33 patients with endocarditis, 20 were cured.

With regard to technique, Dr. Brown said that Gross used a long incision in the third left intercostal space. Harrington adopted a postero-lateral approach. The chest was then opened widely and a better exposure was obtained. In January, 1944, Gross reported 56 cases. In the first fourteen he had exposed the ductus and tied it with two silk ligatures; in some of these cases recurrence took place. Gross then adopted Pearce's technique, using "Cellophane" around the vessel after tying it. There were still some recurrences, so Gross put four clamps over the ductus and divided it and closed the ends by suture. Dr. Brown said that this was the best and most perfect technique of all. It was perhaps more hazardous. Blalock had a different method. He closed the ductus at either end with a purse string suture and then passed mattress sutures through the vessel between the purse string sutures and finally tied an umbilical tie over this. There were no recurrences in Blalock's series. Dr. Brown said that this was a sound technique and he had employed it in his recent cases.

Dr. Brown went on to say that recurrence was a real problem. Major Gebauer, while he was in Australia, had tied the duct in two cases of patent *ductus arteriosus* and assisted at a third operation. In all cases the condition recurred. The third patient had been operated on at the Alfred Hospital, and the condition had recurred after two years. The explanation for this probably lay in the fact that Major Gebauer had operated in five or six cases in Cleveland, in one of which an aneurysm of the ductus developed at the site of ligature and the patient died of its rupture three months later. Major Gebauer decided he had tied his ligature too tightly, and so was cautious not to fall into the error again. He relied too much on "Cellophane" to close the ductus and this proved unsuccessful. Dr. Brown said that recurrence was really only an occasional event. The ligatures must be tied tightly. It was a difficult matter to make a completely occlusive ligature. That was why Blalock's technique was so much better. This allowed very little chance of relapse.

Dr. Brown then said that good anæsthesia was extremely important in these cases. It was not possible to do the work safely without it. Perfect anæsthesia rendered the field immobile. Time did not matter and the operation was made easy. On the other hand, poor anæsthesia made the operation almost impossible. Dr. Brown concluded by saying that Blalock's approach to congenital pulmonary stenosis was interesting; in these cases he had carried out the reverse procedure, creating an artificial *ductus arteriosus*.

Dr. Stokes, in reply, said that the children were presented to show how well they had done since the operation and how quickly they had improved. He was indebted to Dr. Brown for his help with these cases and for his discussion of the subject.

### Hypospadias.

Dr. H. C. Colville presented three male patients, aged respectively eleven, twelve and twenty years, on whom he had operated for hypospadias. In each case the anomaly was of the peno-scrotal type. The whole plastic procedure was divided into three stages, Edmon's technique being used. The first stage consisted of division of the preputial skin. Two incisions were made, both of which divided the dorsal vein but allowed a collateral circulation to develop at the sides. The second operation consisted in straightening the penis and the rudimentary urethra which was present. At this stage the band of tissue holding the penis in the curved portion was severed. The skin flap of the prepuce was then transferred to the ventral surface of the penis and placed in position. The third operation consisted in forming the urethra out of the skin obtained by the second manœuvre. Dr. Colville said great difficulty was experienced in obtaining healing subsequent to the third stage of the operation, and frequently the procedure had to be repeated on more than one occasion. Dr. Colville said that the main methods of promoting healing were the insertion of a self-retaining catheter and the use of penicillin as a dressing locally. With these adjuncts he found that after two or three attempts at the third stage some success was obtained, and his patients were shown to this end. Dr. Colville asked for suggestions to improve his technique and help in these difficult cases.

Dr. H. Douglas Stephens said that hypospadias was the most disappointing of all congenital deformities for the surgeon. The minor varieties of the condition gave little trouble. The greatest difficulty was met with in dealing with the peno-scrotal and perineo-scrotal types. He could recall only one case in which a really good result was obtained. This patient had two fistulæ which were subsequently corrected. Three operations were the minimum required in the third stage. The last patient he had treated needed fourteen operations. Edwards's operation had proved successful in this case. It was a complicated operation and difficult to carry out, but the results were satisfactory. Another operation in the lesser varieties of hypospadias was Dennis Browne's operation. Dr. Stephens said that he had tried Beck's operation, but this had proved a terrible failure. After this operation the patient developed a penis

like a large pear, with all the weight at the distal end. Dr. Stephens had had occasion to remove a large wedge of tissue in one such case. Hamilton Russell's operation was also complex; Russell was repairing some of his results to the end of his days. Macindoe was making a special urethral tube from skin elsewhere and was using this. On one occasion Dr. Stephens said that he had used an appendix. It was often not a difficult matter to make a good front part of the reconstructed urethra, but it was a different matter to join the front and the posterior portions.

DR. WILFRED FORSTER said that it was often urged by parents that the surgeon should attack these conditions too early. Failures were often due to compliance with this wish. Dr. Forster remembered one case at the Children's Hospital in which he had carried out Dennis Browne's operation. This had to be repeated ten times. At the time of the meeting the mother had to hold three chamber-pots in front of the child during micturition; he might be described as having a "watering-can penis". After Ombredanne's operation the patient seemed to have a good urethra, but in thirty-six to forty-eight hours three or four areas of dripping developed. The operation site became œdematous, and the strain due to micturition opened up a number of small fistulæ. Dr. Forster said he had thought of using a suprapubic self-retaining catheter; but even with this dribbling from the urethra occurred and fistulæ developed. He had more recently been passing a catheter *per urethram* soon after the operation and every twelve hours or so afterwards. This left a track open and prevented the leaking out of urine at various points. When this form of treatment was used, the wound healed more readily and an almost perfect urethra was obtained. The catheter need only be passed along the urethra without entering the bladder and then removed, the child passing urine immediately afterwards. Dr. Forster thought the hint might be worth passing on; but not enough of these cases were encountered to allow one to judge the efficacy of such procedures.

*(To be continued.)*

# Naval, Military and Air Force.

## DECORATIONS.

THE following officers of the Australian Army Medical Corps have been created Commanders of the Military Division of the Most Excellent Order of the British Empire "for distinguished services, leadership and devotion to duty": Colonel (Temporary Brigadier) Walter Paton MacCallum, D.S.O., M.C., Lieutenant-Colonel (Temporary Colonel) Charles William Berry Littlejohn, O.B.E., M.C., Colonel (Temporary Brigadier) George Brumfitt Gibb Maitland, D.S.O., D.C.M.

The following have been created Officers of the Military Division of the Most Excellent Order of the British Empire "for highly meritorious service and outstanding devotion to duty": Lieutenant-Colonel Neville Howard Morgan, Major (Temporary Lieutenant-Colonel) Ernest Edward Dunlop, Lieutenant-Colonel Christopher Bagot Sangster.

The following have been created Members of the Military Division of the Most Excellent Order of the British Empire "for highly meritorious service and outstanding ability": Major Bruce Atlee Hunt, Major Bertram William Nairn, Captain (Temporary Major) Ewan Lawrie Corlette, Captain (Temporary Major) Arthur Alexander Moon, Captain John Finch Akeroyd, Captain Desmond James Brennan, Captain Richard Lloyd Cahill, Captain Theodore Godlee.

The following have been created Members of the Military Division of the Most Excellent Order of the British Empire "for meritorious service and outstanding ability": Major (Temporary Lieutenant-Colonel) Lennard Gilmour Travers, Major Harry Vivian Francis, Captain William Kennley Patrick, Major Alfred Kenneth Jones, Major Robert Nicholson McCulloch, Major Ian Webster MacNaught, Major Ewan Murray-Will, Major Colin Campbell Wark, Captain Thomas Victor Walpole.

The following officer has been awarded the military cross for outstanding courage and devotion to duty: Captain Douglas Campbell Pope.

The following have been mentioned in dispatches "for distinguished services in the South-West Pacific Area": Major Clifford Ernest Sawrey, Captain Francis Alfred Leonard Bacon, Captain Geoffrey Patrick Dynon, Captain William Wotherspoon McLaren, Lieutenant-Colonel Norman Menzies Eadie, Captain (Temporary Major) Ewan Lawrie Corlette, Captain (Temporary Major) Arthur Alexander Moon, Captain (Temporary Major) Jack Dudley Morris, Captain Vincent George Bristow, Colonel (Temporary Brigadier) Douglas Munro Salter, Lieutenant-Colonel (Temporary Colonel) William Wettenhall Lempriere, D.S.O., Lieutenant-Colonel Munro Scott Alexander, O.B.E., Lieutenant-Colonel Rex Frederick Allingham Becke, Lieutenant-Colonel Josiah Mark Bonnin, Lieutenant-Colonel Donald William Brummit, Lieutenant-Colonel Sholto John Douglas, Lieutenant-Colonel Alban Harvey Gee, Lieutenant-Colonel John Leicester Holme, Lieutenant-Colonel William Morris Irwin, Lieutenant-Colonel Alan Harding Lendon, Lieutenant-Colonel Joseph Benedict McElhone, Lieutenant-Colonel Frederick Westwood Niesche, Lieutenant-Colonel Mostyn Levi Powell, Lieutenant-Colonel William Dudley Refshauge, O.B.E., Lieutenant-Colonel Richard Stanley Smibert, O.B.E., Lieutenant-Colonel Richard Francis Kirby West, Major (Temporary Lieutenant-Colonel) William Anderson Hugh-Smith, Major John Errol Chandos Aberdeen, Major Alan Sidney Brett, Major Robert Sydney Day, Major William Bernhard Dorsch, Major Lachlan Neil Gollan, Major Reginald Hewgill Hamilton, Major Patrick Gavan Hayes, Major Eric Alfred Hedberg, Major Leslie Parker Hiatt, Major Charles Housten Horsley, Major Wilfred Jabez Lawes, Major James Fishbourne McCulloch, Major Brian Russell Morey, Major George Read, Major Thomas Frederick Rose, Major Maxwell Steven Truscott, Major Donald Watson, Major Terence Keith Smyth Whiting, Major Dudley Clarence Williams, Captain (Temporary Major) Claude James Constable, Captain (Temporary Major) Robert Brown Crawford, Captain (Temporary Major) Frank Christopher Henry Davies, Captain (Temporary Major) Ian Archibald Niel McCallum, Captain (Temporary Major) Walter Gottfried Miller, Captain (Temporary Major) Reginald Ernest Wherrett, Captain Thomas James Arnold, Captain Richard Ulick Bourke, Captain Sydney Edward Bradley, Captain Maxwell William Branagan, Captain Cyril Brasher, Captain Herbert Edward Coxhead, Captain Anton Emil Diethelm, Captain John Eric Graves, Captain David Lindsay Graham, Captain Victor Henry Hegarty, Captain Charles Arthur Holmes, Captain Alan Charles Jeffrey, Captain John Charters Kerridge, Captain James Herschel Nicholls, Captain Roy Lindsay John Park, Captain William Kennley Patrick, Captain William David Pryor, Captain William Matthew James Quinn, Captain Raymond William Sutton Read, Captain Alan Lewis Redmond, Captain Alan Foster Richardson, Captain William Eric Swaney, Captain Owen Upcott Williams.

The following have been mentioned in dispatches "for services rendered whilst prisoners of war": Lieutenant-Colonel William Cotter Burnell Harvey, Lieutenant-Colonel Edmund MacArthur Sheppard, Lieutenant-Colonel John George Glyn White, Major (Temporary Lieutenant-Colonel) Ernest Edward Dunlop, Major Kevin James Fagan, Major Walter Edward Fisher, Major Alan Frank Hobbs, Major Reginald Errol Maffey, Major Robert Graeme Orr, Major Roy Halford Stevens, Captain William Aitken, Captain Claud Leonard Anderson, Captain Douglas Neil Gillies, Captain Roy Markham Mills, Captain Leslie Oswyn Sheridan Poidevin, Captain Charles Rowland Bromley Richards.

# Correspondence.

## THE PRESENT POSITION OF ALLERGY.

SIR: Dr. Bernard Riley in his recent paper, "The Present Position of Allergy", would have been well advised to add "in New South Wales" to the title, for he makes no reference to work done in Victoria and South Australia in the last quarter of a century.

He blames all practising allergists for the dearth of articles on this subject published in Australia, but makes no reference to quite a number of papers by Maxwell, Sutherland and myself written during the inter-war years. Since then no apology has been needed for being too busy to record the results of more recent observations.

Contrary to his experience that the army authorities have frowned on any attempt at investigation and treatment of allergic subjects, the work in this area has been considerable for both army and air force. Dr. Maxwell and I received appointments as consultants in allergy to the army in Victoria and South Australia respectively.

It is difficult to understand how Dr. Riley obtained the impression that the United Kingdom was in a satisfactory position regarding allergy, for this was very far from being the case during my latest visit in 1939.

Only a very small amount of allergic investigation was being carried out, even in London and more recent papers

by the late Sir Arthur Hurst, and others have not reflected any improvement in allergic studies.

As for South Australia, although there is much more to be done in the way of education of students and practitioners, there is at least a widespread appreciation of the value of proper treatment of allergic disorders so that it is difficult to recall a practice from which allergic patients are never referred.

Regarding Dr. Riley's suggestions for improvements, I am in general agreement, but would add that the specialist in allergy should have a solid grounding in general pathology and medicine and gain a higher qualification in medicine, then proceed to the special study of allergy in more than one clinic. In the United States of America a man must spend two years in such clinics before he can be accepted and registered as a specialist. Similar rules here would improve the status of allergists and other specialists, and their influence in educating their colleagues and students would be enhanced.

Yours, etc.,

D. L. BARLOW.

Bank of New South Wales Chambers,
North Terrace and King William Streets,
Adelaide.
March 18, 1947.

## THE FEDERAL GOVERNMENT AND THE MEDICAL PROFESSION.

SIR: There cannot be much doubt that the present Federal Government intends to proceed with the socialization of medicine.

Is it not time that the opinion of the medical profession throughout Australia upon this matter was obtained? This could be done by a *questionnaire*. The opinions of the members of the Association having been determined, then a policy could be drawn up and a plan of campaign adopted to implement that policy. Meanwhile the profession appears to be asleep whilst the thief of their liberty is steadily drawing nigh.

Yours, etc.,

PAUL G. DANE.

111, Collins Street,
Melbourne, C.1,
March 13, 1947.

## THE NEW SOUTH WALES BRANCH COUNCIL ELECTIONS.

SIR: I have just had the privilege, but doubtful pleasure, of completing a ballot paper for the election of the new New South Wales Branch Council of the British Medical Association, and wonder how many more beside myself really do not know who many of the candidates are, still less what are their records and policies in British Medical Association matters.

Out of that, I write to recommend to members that we alter our method of election of the Council at a very early date so as to provide a representation resembling that in Parliament—that, in fact, we elect a medical parliament on a proportional basis, thereby electing men with whom we are in touch and with whose personality and ideas we are quite familiar when it comes to voting day.

At present all is peaceful on the medico-political front, some even are tuning up their fiddles preparing to outdo Nero, but some can see the big black clouds of trouble coming up, and feel the need of the best and most comprehensive organization we can have—one ready for rapid, firm action. We do not want conventions which take time to call up and then only make recommendations, but a standing body whose representatives of each and every facet of medical opinion are able to maintain close contact with the opinion of each of their "electorates": able also to meet them all over the State during the week-end if necessary and to present collectively a mandated Council decision on the Monday.

The plan suggested is to have the profession arranged in so many voting groups, whether geographical or by other community of interest. Thus we will have councillors representing local associations, specialists, women doctors, government medical officers, resident medical officers (and, in wartime, the services), all strictly according to their numbers, every British Medical Association member having one vote only.

This plan has been drawn up in form ready to insert into our constitution and requires only two (special) meetings to pass it, and the approval of the Parent Body: it can be started off any time twenty-five members sign a requisition for it.

Many arguments are put up against this scheme, answers to most of which are ready, but omitted just now to save space. The main opposition for serious consideration is that it would split the profession into political groups and develop true political intrigues: on the contrary, I believe it will bring in many of the profession who are now lax because they feel no intimate interest under the present conditions, and others frankly antagonistic who stand quite aloof from British Medical Association affairs, and will bring about a unity we have not known before.

I should be glad alike to answer questions, to face difficulties and to acknowledge assistance.

Yours, etc.,

CRAWFORD MCKELLAR.

143, Macquarie Street,
Sydney,
March 24, 1947.

## PEPTIC ULCER.

SIR: In THE MEDICAL JOURNAL OF AUSTRALIA, January 11, page 42, I made the statement that patients suffering with hæmatemesis should be admitted to a surgical ward so that those who immediately, or eventually, required a gastrectomy as a life-saving measure, should have it at the earliest possible moment. Those whose condition did not require a gastrectomy could be transferred to a medical ward. Whilst in either ward they could receive such treatment, dietary, fluid replacement, or medicinal, as their condition required.

No one will deny that the most important single feature of the treatment of this accident is the transfusion of blood which, with the various ancillary methods now in use, is persisted with until it becomes obvious that the method is going to fail. The statement that patients suffering with mild hæmatemesis and apparently not requiring any surgical assistance should be admitted to a surgical ward is not unreasonable, as the future course of such cases is not always immediately certain and they can receive adequate treatment whilst in such a ward. The changes in condition that require surgery may arrive either gradually or suddenly. If gradually, it will be predicted easily, and if suddenly, emergency methods can be taken without any unnecessary delay for consultations *et cetera*. The point at which operation should be done is a matter of experience, but the criteria for establishing this are the same in a medical or surgical ward.

In THE MEDICAL JOURNAL OF AUSTRALIA, February 15, 1947, I said that "even with the best transfusion services available, some patients will still die unless a direct attack is made on the bleeding". I insist that this attempt should be made whilst the patient is in such a condition as to lead one to think that there is a reasonable hope of achieving an end, namely, hæmostasis, and not left until it is a desperate try to avoid an imminent disaster. Any machinery such as primary admission to a surgical ward which will facilitate this must be a step in the right direction. In conclusion, I advised Dr. McLean to read your Current Comment, THE MEDICAL JOURNAL OF AUSTRALIA, January 18, 1947.

In reply he has quoted from the same article, but has quoted only so far as suits his argument and stops short at a point beyond which his argument is not supported. Dr. McLean's quotation from Gordon-Taylor is: "These, together with increasing vigilance and enthusiasm on the part of the physicians, have reduced 'medical mortality' to such a low level as to necessitate the greatest caution before surgery is advised." The very next sentence is: "Nevertheless, when properly decided on and expertly carried out, operation will save some patients who would die without it." Gordon-Taylor then advises early consultation between the physician and surgeon and deplores the tendency, becoming less evident, to delay the appeal for surgical advice until the patient is *in extremis*. The "these" mentioned above by Gordon-Taylor are newer methods in assessing the blood loss, the scientific employment of replacement fluids (especially continuous drip transfusion) and earlier recourse to gastric feeding—all of which are as applicable in a surgical ward as in a medical ward.

When a gastric or duodenal hæmorrhage occurs as the result of the erosion of a vessel in the ulcer floor, the pathological process is quite similar to that occurring in a secondary hæmorrhage. It might also be noted again that there is a process of endarteritis occurring in the vessels adjacent to an ulcer which obliterates all but the larger vessels, so that when such an erosion takes place, the bleeding is likely to be severe as the main vessels in these parts are large vessels from which a patient can bleed to

death in a few minutes. In quoting from Mr. Hailes's article on "Secondary Hæmorrhage" (*The Australian and New Zealand Journal of Surgery*, Volume XVI, Number 1, July, 1946, page 39), Dr. McLean has used the same method of partial quotation to give an impression contrary to that desired by the author. Dr. McLean quotes Mr. Hailes as saying that "bleeding from small branches of the main trunk will often cease spontaneously". Mr. Hailes, however, went on to say, "and requires nothing more than a liberal dressing and a firm bandage". The remainder of Mr. Hailes's paragraph is just as applicable to hæmatemesis as to secondary hæmorrhage. He says: "It is not suggested that immediate operation be performed on all cases. The degree of urgency depends on the rapidity of the bleeding and the amount of blood lost. A sudden gush implies an opening in a large vessel and demands urgent action; a continuous trickle may prove just as dangerous, but the need for operative interference may not be immediate."

A surgical mind looks with suspicion on all bleeding, and especially so when the bleeding can become so serious as to threaten life. When, in addition, the bleeding can be hidden until its general effects become apparent, there is ground for even greater suspicion. Therefore, in such a disease, the organization should be such as to make sure that, whilst the lesser cases receive appropriate treatment, the major ones will be subjected to no unnecessary delays. Cases should be classified as serious until it is certain that they are not.

In conclusion, I would like to congratulate Dr. McLean on his excellent results, which, I am sure, are due as much to his "vigilance and enthusiasm" as they are to a special method of transfusion. There may be but few cases of hæmatemesis that require operation, but, when operation saves one that was going to die without it, the surgeon has as much right to rejoice as heaven has over the saving of one sinner.

Yours, etc.,

45, Spring Street,
Melbourne, C.1,
March 21, 1947.

LEO DOYLE.

# Australian Medical Board Proceedings.

## NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act, 1938-1939*, of New South Wales, as duly qualified medical practitioners:

Fink, Siegfried, approved for registration in terms of Section 17 (B) of the *Medical Practitioners Act*, 1938-1945, 115, Macleay Street, Potts Point.

Fisher, Bernard, approved for registration in terms of Section 17 (B) of the *Medical Practitioners Act*, 1938-1945, 183, Macquarie Street, Sydney.

Frank, Alexander, approved for registration in terms of Section 17 (B) of the *Medical Practitioners Act*, 1938-1945, 253, Macquarie Street, Sydney.

Haneman, Samuel, approved for registration in terms of Section 17 (B) of the *Medical Practitioners Act*, 1938-1945, 54, Warner's Avenue, Bondi.

Kassel, Arthur, approved for registration in terms of Section 17 (B) of the *Medical Practitioners Act*, 1938-1945, Molesworth Street, Hillston.

Knossew, Isidor Sigmund, approved for registration in terms of Section 17 (B) of the *Medical Practitioners Act*, 1938-1945, 193, Macquarie Street, Sydney.

Landecker, Hans Martin, approved for registration in terms of Section 17 (B) of the *Medical Practitioners Act*, 1938-1945, 525, New South Head Road, Double Bay.

Leeser, Felix, approved for registration in terms of Section 17 (B) of the *Medical Practitioners Act*, 1938-1945, 243, Elizabeth Street, Sydney.

The following additional qualifications have been registered:

Bond, John Anthony, M.B., 1939 (Univ. Sydney), B.S., 1945 (Univ. Sydney), Newcastle Hospital.

Davies, Geoffrey Wyndham, M.B., 1939 (Univ. Sydney), B.S., 1939 (Univ. Sydney), Wollongong.

Flynn, Michael Richard, M.B., 1919, Ch.M., 1920 (Univ. Sydney), F.R.A.C.S., 1929, M.D., 1931 (Univ. Sydney), 135, Macquarie Street, Sydney.

Furner, Carl Russell, M.B., Ch.M., 1920 (Univ. Sydney), M.R.A.C.P., 1938, Newcastle.

Harbison, Bruce Lister, M.B., B.S., 1929 (Univ. Sydney), F.R.C.S., 1940 (Edinburgh), D.L.O. (R.C.P. and S.), 1943, Hornsby.

Taylor, Helen Margaret, M.B., B.S., 1933 (Univ. Sydney), M.R.C.P., 1940 (London), M.R.A.C.P., 1944, 135, Macquarie Street, Sydney.

## TASMANIA.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners:

Fabian, Ernst, M.D., 1925 (Berlin), St. Helens.

Kudelka, Oscar, M.D., 1928 (Vienna), King Island.

Fletcher, Malcolm Weld, M.B., B.S., 1933 (Univ. Adelaide), M.R.C.P., 1940, M.R.A.C.P., 1946, Launceston.

Gibson, Douglas Barr, M.B., B.S., 1945 (Univ. Adelaide), Launceston.

Devenish-Meares, Molly Constance, M.B., B.S., 1931 (Univ. Sydney), M.R.C.O.G., 1939, Launceston.

Yoffa, Leslie David, M.B., B.S., 1918 (Univ. Melbourne), Campbell Town.

Gibb, Herbert Ian, M.B., B.S., 1925 (Univ. Melbourne), Hobart.

## QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts*, 1939, of Queensland, as duly qualified medical practitioners:

Wilcox, George Griffiths, M.B., 1946 (Univ. Sydney), *General Hospital, Brisbane.*

Schmidt, Eric Egmont, M.B., B.S., 1945 (Univ. Sydney), Barolin Street, Bundaberg.

Fitzhardinge, Julie Maude, M.B., B.S., 1946 (Univ. Sydney), General Hospital, Brisbane.

Hede, John Allen, M.B., B.S., 1941 (Univ. Melbourne), *Brisbane Mental Hospital, Goodna.*

Aston, Clifford Ellis, M.R.C.S. (England), L.R.C.P. (London), 1937, M.B., B.S. (London), 1938, c.o. N. *Keyes, Esq., Norman Avenue, Lutwyche, Brisbane.*

Wright, Stanley James, M.B., B.S., 1943 (Univ. Sydney), District Hospital, Atherton.

# Obituary.

## LOUIS EDWARD BARNETT.

WE are indebted to Sir Henry Newland for the following appreciation of the late Sir Louis Barnett, of Dunedin, whose death occurred recently.

In the days to come, the name of Sir Louis Barnett will always have two associations, the foundation of the Royal Australasian College of Surgeons and the Hydatid Registry established by that body. His fame in the annals of Australasian surgery is therefore secure. I had the honour of being President of the Surgical Section of the Eleventh Session of the Australasian Medical Congress held at Brisbane in August, 1920. At the meeting of the Surgical Section held on August 26, attention was directed towards the question of the establishment of an association or fellowship of Australasian surgeons. This was the outcome of a letter addressed by Professor L. E. Barnett, C.M.G., F.R.C.S., Professor of Surgery, University of Otago, to the Honorary Secretary of the Surgical Section of Congress. The writer had suggested that action might be taken somewhat on the lines of the American Fellowship, in order to obtain a higher grade of efficiency in surgical work, and to provide for the bestowal of some hall-mark on the efficient. Sir Louis's proposal was rejected on two grounds: (i) that the creation of such a body would seriously interfere with the surgical sections of the State Branches of the British Medical Association; (ii) that the universities of Australia with medical schools offered a Mastership of Surgery to those who were competent. The rejection was but temporary, and eventually the Royal Australasian College of Surgeons, to the advantage of surgery in Australia and New Zealand, was founded on a secure basis. Although the name of Sir Louis Barnett is recorded with others as one of the founders of the Royal Australasian College of Surgeons, it is but just to regard him as the prime founder of the College.

The prevalence of hydatid disease in Australasia prompted him to secure the creation of a hydatid registry by the

Royal Australasian College of Surgeons. By his foresight, valuable records have been gained which would otherwise have been lost to the world of medicine.

Australasian surgery has good reason to revere the name of Sir Louis Barnett, whose services to surgery were characterized by a humility, which is one of the stamps of the true surgeon.

### WILLIAM CARLYLE WILSON.

We regret to announce the death of Dr. William Carlyle Wilson, which occurred on March 24, 1947, at Eastwood, New South Wales.

### LACHLAN GOLLAN.

We regret to announce the death of Dr. Lachlan Gollan, which occurred on March 26, 1947, at Ulverstone, Tasmania.

### FREDERICK WILLIAM DEAN COLLIER.

We regret to announce the death of Dr. Frederick William Dean Collier, which occurred on March 29, 1947, at Sydney.

### GUY DIXON MENZIES.

We regret to announce the death of Dr. Guy Dixon Menzies, which occurred on March 30, 1947, at Drummoyne, New South Wales.

## Nominations and Elections.

The undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Stephens (*née* Yeomans), Norma Clyde, M.B., B.S., 1946 (Univ. Sydney), 57, Yarranabbe Road, Edgecliff.

Solling, Mena Gwendolyn, M.B., 1939 (Univ. Sydney), Royal Alexandra Hospital for Children, Camperdown.

Cathers, Wilbur Jackson, M.B., B.S., 1944 (Univ. Sydney), 39, French's Road, Willoughby.

Havyatt, Betty Carlisle, M.B., B.S., 1946 (Univ. Sydney), 3, Bellevue Park Road, Bellevue Hill.

Chesterfield-Evans, Hugh Harvey, M.B., B.S., 1946 (Univ. Sydney), 36, Bulkara Road, Bellevue Hill.

## Medical Appointments.

Dr. H. J. Edelman has been appointed honorary clinical assistant to the medical section of the Royal Adelaide Hospital, Adelaide.

Dr. W. H. J. Cole has been appointed medical officer of health to the Nannup Road Board, Western Australia, under the provisions of *The Health Act*, 1911-1944.

Dr. R. H. Minton Connell has been appointed medical officer of health to the Mount Magnet District Road Board, Western Australia, under the provisions of *The Health Act*, 1911-1944.

## Books Received.

"Principles of Anatomy and Physiology for Physical Training Instructors in the Royal Air Force": 1946. London: His Majesty's Stationery Office. 9½" × 6", pp. 186, with many illustrations. Price: 7s. 6d.

"The Eye Manifestations of Internal Diseases", by I. S. Tassman, M.D.; Second Edition; 1946. St. Louis: The C. V. Mosby Company; Melbourne: W. Ramsay (Surgical), Proprietary, Limited. 9½" × 6¾", pp. 614, with many illustrations, some of them coloured. Price: 75s.

"Crusts and Crusades: Tales of Bygone Days", by the Right Honourable W. M. Hughes, C.H., K.C., M.P.; 1947. Sydney, London: Angus and Robertson Limited. 8½" × 5½", pp. 246. Price: 8s. 6d.

"The Preservation of Proteins by Drying: With Special Reference to the Production of Dried Human Serum and Plasma for Transfusion", by R. I. N. Greaves; Medical Research Council of the Privy Council, Special Report Series Number 258; 1946. London: His Majesty's Stationery Office. 9½" × 6", pp. 82, with illustrations. Price: 2s.

"The 1946 Year Book of the Eye, Ear, Nose and Throat"; the Eye, by L. Bothman, M.D., the Ear, Nose and Throat, by S. J. Crowe, M.D., with the collaboration of E. M. Hagens, M.D.; 1946. Chicago: The Year Book Publishers, Incorporated. 7" × 4¾", pp. 544, with illustrations. Price: $3.75.

## Diary for the Month.

April 8.—Tasmanian Branch, B.M.A.: Ordinary Meeting.
April 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee. Organization and Science Committee.
April 10.—South Australian Branch, B.M.A.: Council Meeting.
April 11.—Queensland Branch, B.M.A.: Council Meeting.
April 14.—Victorian Branch, B.M.A.: Finance Meeting.
April 15.—New South Wales Branch, B.M.A.: Medical Politics Committee.

## Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

*New South Wales Branch* (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

*Victorian Branch* (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

*Queensland Branch* (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute; Brisbane City Council (Medical Officer of Health). Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

*South Australian Branch* (Honorary Secretary. 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

*Western Australian Branch* (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

## Editorial Notices.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to The Medical Journal of Australia alone, unless the contrary be stated.

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